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Delayed Onset Muscle Soreness

Treatment Strategies and Performance Factors

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Abstract

Delayed onset muscle soreness (DOMS) is a familiar experience for the elite or novice athlete. Symptoms can range from muscle tenderness to severe debilitating pain. The mechanisms, treatment strategies, and impact on athletic performance remain uncertain, despite the high incidence of DOMS. DOMS is most prevalent at the beginning of the sporting season when athletes are returning to training following a period of reduced activity. DOMS is also common when athletes are first introduced to certain types of activities regardless of the time of year. Eccentric activities induce micro-injury at a greater frequency and severity than other types of muscle actions. The intensity and duration of exercise are also important factors in DOMS onset. Up to six hypothesised theories have been proposed for the mechanism of DOMS, namely: lactic acid, muscle spasm,

connective tissue damage, muscle damage, inflammation and the enzyme efflux theories. However, an integration of two or more theories is likely to explain muscle soreness. DOMS can affect athletic performance by causing a reduction in joint range of motion, shock attenuation and peak torque. Alterations in muscle sequencing and recruitment patterns may also occur, causing unaccustomed stress to be placed on muscle ligaments and tendons. These compensatory mechanisms may increase the risk of further injury if a premature return to sport is attempted.

A number of treatment strategies have been introduced to help alleviate the severity of DOMS and to restore the maximal function of the muscles as rapidly as possible. Nonsteroidal anti-inflammatory drugs have demonstrated dosage-dependent effects that may also be influenced by the time of administration. Similarly, massage has shown varying results that may be attributed to the time of massage application and the type of massage technique used. Cryotherapy, stretching, homeopathy, ultrasound and electrical current modalities have demonstrated no effect on the alleviation of muscle soreness or other DOMS symptoms. Exercise is the most effective means of alleviating pain during DOMS, however the analgesic effect is also temporary. Athletes who must train on a daily basis should be encouraged to reduce the intensity and duration of exercise for 1–2 days following intense DOMS-inducing exercise. Alternatively, exercises targeting less affected body parts should be encouraged in order to allow the most affected muscle groups to recover. Eccentric exercises or novel activities should be introduced progressively over a period of 1 or 2 weeks at the beginning of, or during, the sporting season in order to reduce the level of physical impairment and/or training disruption. There are still many unanswered questions relating to DOMS, and many potential areas for future research.

Following unaccustomed physical activity, a sensation of discomfort, predominantly within the skeletal muscle, may be experienced in the elite or novice athlete. The intensity of discomfort increases within the first 24 hours following cessation of exercise, peaks between 24 and 72 hours, subsides and eventually disappears by 5–7 days post-exercise.[1-5] This exercise-induced phenomenon is referred to as delayed onset muscle soreness (DOMS) and is perhaps one of the most common and recurrent forms of sports injury.

Numerous studies have attempted to identify prevention strategies for DOMS.[5-22] The absence of a known preventative measure and the diverse range of treatment techniques available are largely due to the lack of understanding surrounding the exact mechanisms of DOMS.[17] Currently, as many as six hypothesised theories have been proposed as potential aetiological explanations for the muscular pathology.[2,5,17,23,24] It is not within the scope of this paper to address in detail the proposed theories of DOMS. This has been well reviewed by other investigators.[2,5,17,25] Rather, the aim of this paper is to discuss the impact of muscular soreness on athletic performance and to review the current research on the treatment techniques and management strategies of DOMS.

1. Literature Reviewed

Literature was located using two computer databases (Medline and SPORT Discus) in addition to manual journal searches. The computer databases provided access to biomedical and sports-oriented journals, serial publications, books, theses, conference papers and related research published since 1948. The keywords used included: muscle soreness, DOMS, delayed onset muscle soreness, muscle injury, muscle strain, injury prevention and ec-

centric exercise. Excluding articles that were not published in English and/or in scientific journals, refined the literature searches. Articles that focused on the psychological effects of DOMS, or the effect of DOMS in special populations, were not included in the review. The criteria for inclusion were:

- the paper must have addressed at least one mechanism and/or proposed treatment of prevention for DOMS
- the paper must have used normal, healthy participants. Age, sex, fitness differences were not an excluding factor
- DOMS may have been discussed in relation to other forms of muscle injury, e.g. muscle strain, cramp
- the paper may have been a review of previous research.

2. Definition of Delayed Onset Muscle Soreness (DOMS)

DOMS is classified as a type I muscle strain injury[17,26] and presents with tenderness or stiffness to palpation and/or movement.[17] Although the pathology associated with DOMS is usually subclinical,[27] the sensations experienced with this injury can vary from slight muscle stiffness, which rapidly disappears during daily routine activities, to severe debilitating pain which restricts movement.

Tenderness is concentrated in the distal portion of the muscle[2,24,27-30] and becomes progressively diffuse by 24–48 hours post exercise.[24] This localisation of pain can be attributed to a high concentration of muscle pain receptors in the connective tissue of the myotendinous region.[31] The myotendinous junction is characterised by a membrane which is continuous, extensively folded and interdigitated with the muscle cells.[29] The oblique arrangement of the muscle fibres just prior to the myotendinous junction reduces their ability to withstand high tensile forces.[29,32,33] As a result, the contractile element of the muscle fibres in the myotendinous junction is vulnerable to microscopic damage.

DOMS is usually associated with unfamiliar, high-force muscular work and is precipitated by eccentric actions.[27] Eccentric activity is characterised by an elongation of the muscle during simultaneous contraction. Thus, if the external load exceeds the muscle's ability to actively resist the load, the muscle is forced to lengthen and active tension is generated.[34] Cross bridges formed during eccentric actions must also be separated with greater force due to the disruption of the actin-myosin bonds prior to relaxation.[34] As a result, greater tension per active motor unit is developed and there is an increased risk of injury to the vulnerable myotendinous junction. Researchers investigating the mechanisms of DOMS have induced muscle soreness using exercise protocols consisting of predominantly eccentric activity, i.e. downhill running,[35-42] resisted cycling,[43-46] ballistic stretching,[47] isokinetic dynamometry,[14,17,20,32,48-61] stepping[7,8,25,62-64] and/or eccentric resistance exercise.

3. Mechanisms of DOMS

A number of theories have been proposed to explain the pain stimulus associated with DOMS including: lactic acid, muscle spasm, connective tissue damage, muscle damage, inflammation, enzyme efflux theories and other proposed models.[5,17] It is beyond the scope of this review to provide a detailed discussion of each theory. Rather, it is the intention of the authors to provide a brief overview of the mechanisms and how they interrelate so that a basic understanding of the possible mechanism(s) of soreness perception may be obtained.

The lactic acid theory is based on the assumption that lactic acid continues to be produced following exercise cessation. For the lay public, the accumulation of toxic metabolic waste product is thought to cause a noxious stimulus and the perception of pain at a delayed stage.[2,17,22] However, this theory has largely been rejected as the higher degree of metabolism associated with concentric muscle contractions have failed to result in similar sensations of delayed soreness.[65] In addition, lactic acid levels return to pre-exercise levels within 1 hour following exercise and blood lactate levels measured before, during and sporadically up to 72 hours after level and downhill running have failed to show a relation-

ship between lactic acid levels and soreness ratings.[42] Therefore, lactic acid may contribute to the acute pain associated with fatigue following intense exercise, however, it can not be attributed to the delayed pain that is experienced 24–48 hours post-exercise.[66]

The muscle spasm theory[67] was introduced following the observation of increased levels of resting muscle activity after eccentric exercise.[17,68,69] It was proposed that an increased resting muscle activation indicated a tonic localised spasm of motor units. This was thought to lead to a compression of local blood vessels, ischaemia and the accumulation of pain substances. In turn this initiated a 'vicious cycle'[70] as further stimulation of pain nerve endings caused further reflex muscle spasms and prolonged ischaemic conditions.[68] However, investigations using both bipolar and unipolar electromyography (EMG) have been inconclusive with some studies showing no increase in EMG activity in sore muscles,[1,25,71] whilst others have observed an increase in EMG, but no relation between its magnitude and the perception of soreness.[68] The use of bipolar and unipolar electrode techniques remains controversial with some researchers arguing that the former method lacks the sensitivity to record the electrical activity in sore muscles,[72] whilst others have argued the contrary.[25]

The connective tissue damage theory examines the role of the connective tissue that forms sheaths around bundles of muscle fibres. The content and composition of connective tissue differs between muscle fibre types. Type I (slow twitch) fibres display a more robust structure than type II (fast twitch) fibres. Subsequently fast twitch fibres may demonstrate an increased susceptibility to stretch-induced injury[34] and excessive strain of the connective tissue may lead to muscle soreness.[73] Measurements of urine excretion hydroxyproline (HP) and hydroxylysine (HL) following exercise have been examined to provide support for this theory.[74] HP and HL amino acids are a component of mature collagen and their presence in urine is the result of collagen degradation either by overuse or strain damage.[34] However, HP and HL excretion can reflect either increased collagen synthesis or its degradation. Subsequently, the specific mechanism leading to an increase in HP and HL remains uncertain.

The muscle damage theory, first proposed by Hough,[73] focuses on the disruption of the contractile component of the muscle tissue, particularly at the level of the z-line, following eccentric exercise.[2,4,71,75-78] The characteristic microscopic lesion is a broadening, smearing or even total myofibrillar disruption of the z-line,[79] in addition to more widespread disruption of sarcomere architecture.[39] This damage is the result of increased tension per unit area caused by a reduction in active motor units during eccentric actions.[2] Mechanical disruption to the structural elements is increased, particularly amongst the type II fibres which have the narrowest and weakest z-lines. Nociceptors situated in the muscle connective tissue and in the region of the arterioles, capillaries and the musculotendinous junction are also stimulated leading to the sensation of pain. Blood enzymes have been measured post-exercise to support this theory. Creatine kinase (CK) is considered a reliable indicator of muscle membrane permeability as this enzyme is found exclusively within skeletal and cardiac muscle.[5] Thus, disruption of the z-lines and damage to the sarcolemma will enable the diffusion of soluble muscle enzymes, such as CK, into the interstitial fluid. In normal resting conditions, plasma CK is approximately 100 IU/L.[71] However, following eccentric exercise, circulating levels of CK have been known to rise to 40 000 IU/L,[71] indicating a significant increase in the permeability of the muscle cell membranes following z-line disruption.[39,69,80,81] Yet there is a clear discrepancy between the time of peak serum CK levels and peak muscle soreness (up to 5 days).[39,43,45,54,59,71,82,83] As a result, the muscle damage theory can only be accepted as a partial explanation for the onset of DOMS.

The inflammation theory is based on the finding that aspects of the inflammatory response, i.e. oedema formation and inflammatory cell infiltration, are evident following repetitive eccentric muscle action.[22,45,84] Muscle fibres contain proteolytic enzymes which initiate the degradation of lipid and

protein structures of cells following injury. This rapid breakdown of damaged muscle fibres and connective tissue in addition to the accumulation of bradykinin, histamine and prostaglandins, attracts monocytes and neutrophils to the injury site.[62] This is followed by an influx of protein-rich fluid (exudate) into the muscle via the increased permeability of small blood vessels following eccentric exercise.[84] Ultimately, an osmotic pressure is exerted and pain is produced if group IV sensory neurons are activated.[32] However, only peak oedema levels (as measured by limb volume and girth) appear to coincide with peak muscle soreness;[14,17] the time course of inflammatory cell infiltration is less coincidental.[4,41] This may explain why some authors have chosen to address this mechanism simply as the Tissue Fluid Theory.[17] Nonetheless, Smith[84] and Armstrong[2] have both argued that monocytes, which convert into macrophages, accumulate at the injury site and produce substances which sensitise the type III and IV nerve endings within 24–48 hours. Whether oedema formation, as well as inflammatory cell infiltration, are mechanisms responsible for DOMS remains controversial.

The enzyme efflux theory proposed by Gulick and Kimura[17] is based on the assumption that calcium, which is normally stored in the sarcoplasmic reticulum, accumulates in injured muscles following sarcolemmal damage.[2] This is thought to lead to an inhibition of cellular respiration at the mitochondrial level causing adenosine triphosphate (ATP) regeneration, which is required for the active transport of calcium back into the sarcoplasmic reticulum, to slow. In addition, calcium accumulation is also thought to activate proteases and phospholipases, thus causing further injury to the sarcolemma with the production of leukotrienes and prostaglandins.[2,81] As a result, muscle protein degeneration at the weakened z-lines increases and the chemical stimulation of pain nerve endings occurs.

The general consensus amongst researchers is that a single theory cannot explain the onset of DOMS. As a result, some researchers have proposed unique sequences of events in order to explain the DOMS phenomenon.[2,85] These models integrate aspects from the above theories and start with the assumption that high tensile forces, associated with eccentric exercise, damage muscle tissue and connective tissue initially. This is followed by an acute inflammatory response consisting of oedema formation and inflammatory cell infiltration. An integration of the models proposed by Armstrong,[2,81] Smith[84] and Smith and Jackson[85] can be described as follows:

- High tensile forces produced during eccentric muscle activity cause disruption of structural proteins in muscle fibres, particularly at the weakened z-lines. This is accompanied by excessive strain of the connective tissue at the myotendinous junction and surrounding muscle fibres (connective tissue damage theory and muscle damage theory).
- Damage to the sarcolemma results in the accumulation of calcium that inhibits cellular respiration. ATP production is hindered and calcium homeostasis is disturbed. High calcium concentrations activate calcium-dependent proteolytic enzymes that degrade the z-line of sarcomeres, troponin and tropomyosin (enzyme efflux theory).
- Within a few hours there is a significant elevation in circulating neutrophils (inflammation theory).
- Intracellular components and markers of connective tissue damage and muscle damage (e.g. HP and CK) diffuse into the plasma and interstitium. These substances serve to attract monocytes between 6–12 hours that in turn convert to macrophages. Mast cells and histamine production are activated (inflammation theory). Within hours there is a significant elevation in circulating neutrophils at the injury site (inflammation theory).
- Monocytes/macrophages peak in number at 48 hours. Upon exposure to the inflammatory environment, macrophages produce prostaglandin (PGE_2) that sensitises type III and IV nerve endings to mechanical, chemical or thermal stimulation (inflammation theory).
- The accumulation of histamine, potassium and kinins from active phagocytosis and cellular necrosis in addition to elevated pressure from tissue

oedema and increased local temperature could then activate nociceptors within the muscle fibres and the muscle tendon junction (inflammation theory).
- These events lead to the sensation of DOMS. Soreness may be increased with movement as the increased intramuscular pressure creates a mechanical stimulus for pain receptors already sensitised by PGE_2.

At present, the above sequence of events remains hypothetical. It is evident that further research is warranted to validate the biochemical and cellular events that occur leading up to the onset of muscle soreness.

4. Impact of DOMS on Athletic Performance

In addition to muscle soreness, structural damage to muscle and connective tissue incurred during eccentric activity may result in alterations to muscle function and joint mechanics.[86] For the elite athlete, these adaptations, together with any other compensatory mechanisms for soreness relief, may cause significant alterations to, and a reduction in, performance and/or a less than optimal training intensity.

4.1 Perception of Functional Impairment

Past research has reported independent changes in joint kinematics and muscle function during DOMS.[15,77,87,88] However, minimal research has focused on an individual's perception of his/her physical impairment. Impairment can be defined as an objectively measurable alteration of anatomical, physiological or psychological status of the human being.[89] Examples of impairment include a decreased range of joint motion, decreased strength or abnormal electromyographical patterns. A restriction or the lack of ability to perform an activity or function within the range considered normal for an individual can also be described as a functional limitation.[89] This may have important implications during periods of muscle soreness as an athlete's incorrect perception of his/her temporary impairment may lead to an increased risk of injury. Saxton et al.[90] observed a reduced joint proprioception and an overestimation of force production in 12 participants (six male, six female) following 50 maximal eccentric muscle actions of the elbow flexors. Despite being confident that the force sustained in the experimental arm was equal to that being supported in the control arm, participants were consistently producing 35% of the force of the experimental arm. Participants were also unable to match the reference joint angle of the control arm following eccentric exercise; instead constant overshooting of the reference angle was observed. These findings provide preliminary evidence to suggest that neuromuscular function is impaired by the performance of unaccustomed eccentric exercise that induces muscle soreness.[90] Since proprioception is also dependent on afferent receptors located in the skeletal muscle and tendons, this also suggests that muscle damage must have occurred during eccentric exercise.

4.2 Joint Kinematics

Kinematic analyses of running gait following DOMS inducement have revealed varied findings. Hamill et al.[91] reported statistically significant differences in maximum ankle dorsiflexion and plantar flexion during the support phase, a reduction in maximum knee joint flexion in both swing and support phases, and a reduction in maximum hip flexion at touch down in ten females following 30 minutes of downhill running. It was hypothesised that these changes were a compensatory response to the reduced range of motion in the muscle group most affected by DOMS (i.e. quadriceps). The reduced ability of the knee and hip joints to attenuate shock (as a result of the reduced range of motion of the quadriceps muscle) was also thought to be compensated for by the ankle joint and was observed as increased dorsiflexion during support (23.1–25.3°). Supporting this theory, Goff et al.[92] used accelerometry data to report an increased attenuation of shock in the legs and a reduced attenuation at the head in nine male runners during level running 24–120 hours following 30 minutes of downhill running. The researchers attributed these findings to the body's ability to adapt and protect the leg from

injury during running. In contrast, Harris et al.[88] found no significant differences in hip or ankle range of motion but reported a shortened stride length and a reduced excursion of the lower limb at various running speeds.

Significant reductions in joint range of motion have been reported by other researchers[22,55,93-96] following repetitive eccentric resistance exercise[55] and maximal voluntary eccentric contractions of the elbow flexors.[97] Jones et al.[97] attributed their findings to a shortening of the non-contractile elements (i.e. muscle connective tissue), which acts in parallel with the muscle fibres. In one participant, up to 70N of force was required to forcibly straighten the arm. The reduced joint range of motion was not accompanied by a stretch receptor-induced increase in electrical activity in the primary muscles. Howell et al.[96] also failed to demonstrate any spontaneous activity in the elbow flexors following repetitive eccentric lowering of a weight (60–80% of maximum isometric load). As a result, the reduced joint range of motion/stiffness has not been attributed to increased muscle activity, but to significant increases in the swelling of affected tissues, particularly within the perimuscular connective tissue and regions of the myotendinous junction.[96] This swelling is characteristic of an acute inflammatory response to muscle damage or injury.[22,45,84]

4.3 Strength and Power

Significant reductions in strength and power parameters during DOMS have been documented by numerous researchers.[15,37,54,55,62,63,80,98-100] These reductions are most notable in eccentric muscle actions, although concentric and isometric strength losses have also been reported.[15,37,54,55,62,63,80,98-100] Peak torque deficits are most pronounced 24–48 hours following DOMS-inducing exercise and are more profound and persistent during eccentric testing.[47] The duration of strength reduction is also greater following eccentric activity and may require up to 8–10 days to return to normal baseline levels,[101] whilst concentric and isometric strength has been shown to recover within 4 days. Evans et al.[102] observed significant decreases in isokinetic eccentric peak torque of the elbow flexors at 0 hours (43.5%), 24 hours (38.8%) and 48 hours (−32%) following repetitive eccentric contractions using the Biodex isokinetic dynamometer (60°/sec). By 336 hours (14 days), eccentric peak torque had returned to normal. Other researchers have reported a delayed return of eccentric peak torque of the elbow flexors following isokinetic eccentric exercise, with eccentric peak torque remaining at a value 15% less than initial peak torque until 7 days post-DOMS inducement.[100] Similar trends have also been observed with respect to the lower limb; Eston et al.[98] measured isokinetic, eccentric and concentric knee extension peak torque values of the dominant leg at 0.52 and 2.83 radians/sec 2, 4 and 7 days following DOMS-inducing isokinetic exercise. The results showed an immediate post-exercise loss in peak torque for both modes of exercise at slow and fast velocities up to day 4, and a return to normal levels between days 4–7. Significant reductions in concentric and eccentric peak torque in the lower limb 48 hours following DOMS-inducing exercise have also been reported in other studies.[62,63]

Yet despite these findings, the duration of strength recovery still remains equivocal. As these studies have shown, many researchers have failed to collect data at regular intervals over the 48-hour period or have only recorded on selected days following DOMS inducement. As a result, the time at which peak torque returns to normal levels remains uncertain. This may have important implications for the athlete as an alteration in the strength ratio of agonist to antagonist muscle groups may contribute to an increased risk of injury.[103]

4.4 Altered Recruitment Patterns

Muscular injury can often be characterised by the presence of muscle dysfunction, a term used to describe unusual patterns of muscle recruitment during a prescribed set of movements.[104] Any injury to the muscle or connective tissue during eccentric exercise may lead to changes in recruitment patterns or changes in the temporal sequencing of muscle activation patterns. Such alterations are significant as they can result in changes in muscle co-ordination

and segment motion. Although this area has not been well examined in past DOMS research, studies that have examined muscle activity following eccentric exercise have shown promising findings. Miles et al.[105] reported a lengthening of the triphasic EMG pattern of the elbow musculature during elbow flexion in ten non-weight trained females following 50 maximal velocity eccentric repetitions of elbow flexion through 0–90°. Specifically, numerous indicators of altered neuromuscular control were recorded, including lengthening of motor time, time to biceps peak EMG, time to peak velocity and slowing of peak velocity. These responses persisted for up to 5 days and were attributed to muscular fatigue and dysfunction at the level of the muscle due to the high force eccentric exercise. The slowing of peak velocity and the lengthening of the triphasic pattern were thought to be related to the selective damage of fast twitch fibres during high-force eccentric exercise. Alterations in the temporal sequencing of muscles may also be due to a lengthening of the electromechanical delay. The electromechanical delay refers to the time lag between the onset of myoelectrical activity and tension development in a muscle contraction and includes the time required for the conduction of action potentials, the release of calcium, the formation of cross bridges, the development of tension, and the stretching of the series elastic components.[106] Zhou et al.[106,107] reported an approximately 20 msec elongation of the electromechanical delay after fatiguing exercise of an isometric nature, and attributed the elongation to either impaired muscle conductive, contractile or elastic properties or an increased muscle temperature. As similar damage has been reported following eccentric exercise, an increase in the electromechanical delay and alterations in the temporal sequencing of muscles is also conceivable during DOMS. This in turn may affect the temporal sequencing and co-ordination of muscles during functional activity. However, further research is still needed to confirm these assumptions.

An alternative means of assessing muscular dysfunction using EMG techniques is to analyse the ratio of EMG amplitude between pairs of muscles that perform similar or opposing functions.[104] This method of EMG assessment has been successful in the diagnosis of patellofemoral pain[108] and back and neck muscular dysfunction,[104] and involves the identification of compensatory increases in EMG activity (hyperactivity) in areas of the muscle which are uninjured or in other muscle groups (synergists or antagonists). Whether this method can also be used to identify muscular dysfunction during DOMS warrants further investigation.

4.5 Injury Risk Factors

Although DOMS is presently a sub-clinical injury that is privately tolerated, there is certainly potential for DOMS to lead to more costly and debilitating injury. Due to the increasing emphasis on health and fitness promotion, it is becoming commonplace for individuals to continue exercising during periods of intense muscle soreness. Those striving to improve or to maintain levels of fitness or performance regularly adhere to the 'no pain, no gain' philosophy. As a result, the first instinct is to 'work through the pain' as opposed to resting the affected areas.[47] The potential impact of such behaviour could be detrimental to weakened tissue and to any unaccustomed tissue structures that are forced to compensate during the period of functional deficiency post-DOMS inducement. The following risk factors should be considered prior to returning to sport:

- The cushioning effect from a full range of joint motion during landings in running, or landing after a jump, may be reduced during DOMS. The reduced capacity to efficiently absorb shock at impact places the joints and tissue structures under unaccustomed loading.[47] To compensate, increased shock absorption will occur at other joints causing unaccustomed strain to be placed on other muscles, joints, ligaments and tendons.
- Changes in co-ordination and segment motion may result from alterations in muscle sequencing caused by DOMS.[104] This may lead to unaccustomed strain being placed on muscles, ligaments and tendons during functional activity.
- A reduction in force output by an injured part of a muscle may lead to compensatory recruitment

from an uninjured area of a muscle, or from other muscles.[104] This leads to a marked increase in EMG activity (hyperactivity), altered EMG ratios and increased force production of the compensating muscles, causing unfamiliar stress to be placed on the compensating muscle groups.

- A reduction in strength and power during intense muscle soreness may lead to an individual working at a higher intensity than to which they are normally accustomed.[47] An individual prescribed to work at a specific percentage of one repetition maximum for example, may continue to train at a pre-determined intensity during DOMS. However, as a result of damaged and weakened muscle fibres following eccentric activity, the intensity is now relatively higher thus increasing the risk of further injury.
- An increased incidence of injury may also be observed if there is an alteration in the strength ratio of agonist and antagonist muscle groups.[103] Whether DOMS can lead to significant changes in the strength ratio of opposing muscle groups has not been extensively researched.
- An inaccurate perception of impairment may lead to an individual returning to high intensity activity before adequate recovery.

5. Treatment and Management Strategies for DOMS

The proposed mechanisms of DOMS have allowed researchers to investigate various treatment strategies aimed at alleviating the symptoms of DOMS, restoring the maximal function of the muscles as rapidly as possible and/or reducing the magnitude of the initial injury.[17] Treatment strategies have been administered either prophylactically as a preventative measure and/or therapeutically as a treatment measure. Treatment strategies have included cryotherapy, stretching, anti-inflammatory drugs, ultrasound, electrical current techniques, homeopathy, massage, compression, hyperbaric oxygen and exercise.

5.1 Cryotherapy

The initial treatment recommended for traumatic soft tissue injuries is R.I.C.E (rest, ice, compression and elevation). The superficial application of ice results in changes in skin, subcutaneous, intramuscular and joint temperatures.[109] A decrease in tissue temperature stimulates cutaneous receptors to excite the sympathetic adrenergic fibres causing the constriction of local arterioles and venules. This results in a reduction of swelling and a decreased rate of metabolism which in turn reduces the inflammatory response, vascular permeability and the formation of oedema.[15,109] However, studies to date have shown little or no attenuation of the magnitude of muscle soreness or the facilitation of its recovery following the application of cryotherapy.[17,21,110-113] Paddon-Jones and Quigley[15] observed DOMS in eight resistance-trained males after performing 64 eccentric actions of the elbow flexors. Immediately following the completion of the eccentric exercise session the experimental arm was subjected to five 20-minute ice water immersions, with 60-minutes recovery between each immersion. The results showed no significant differences in muscle soreness, isometric torque, isokinetic torque or limb volume between the experimental arm and the control arm at baseline or following ice immersion. Similarly, no differences in the perception of muscle soreness were reported following studies using single ice massage applications of 15 minutes duration either immediately, 24h or 48h post-exercise;[114] 20 minutes duration immediately post-exercise[93] or following immersion of the experimental limb in an ice bath 25 minutes prior to exercise.[115] The lack of significant findings can not be attributed to the training status of the participants as both trained[15] and untrained participants[93] were observed. This contradicts beliefs that trained individuals are more resistant to eccentric exercise-induced injury, more resistant to muscle damage and thus more likely to demonstrate a positive response to cryotherapy.[15] Variations in the frequency of cold application (either in multiple or single applications) and the timing of application (either pre- or post-exercise) also appear to have minimal effect on minimising muscle soreness and

muscle function. Whilst it is possible that an increased number of applications (more than five) and an increased or decreased duration of ice application could be employed, their effect remains equivocal. These protocols would also be impractical for the individual to administer and applications of more than 20 minutes duration have not been recommended (20 minutes is generally recommended in order to effectively lower deep muscle temperature, whilst preventing nerve injury).[116] Thus, cold application, other than its analgesic effect, provides little benefit in the prevention and treatment of DOMS. This contrasts with the effectiveness of cryotherapy in acute traumatic injury and may indicate that a different or a much smaller magnitude of inflammatory response occurs during DOMS.

5.2 Stretching

Static stretching, pre or post-exercise, has been recommended[53] as a preventative measure of DOMS as it is thought to relieve the muscle spasm described in de Vries' muscle spasm theory. Bobbert et al.[68] later proposed that the static stretching of sore muscles post-exercise could also force the dispersion of oedema which accumulates following tissue damage. Repeated and held stretching reduces the tension on the muscle-tendon unit at any given length. This visco-elastic behaviour of the muscle-tendon unit implies a combination of viscous properties, where deformation is rate dependent, and elastic properties, where deformation is load dependent.[117] A visco-elastic material when held at the same tension will increase in length over time (creep). Alternatively, if the visco-elastic material is stretched to a new length and held constant, it will decline in tension over time (stress-relaxation). This visco-elastic behaviour could be beneficial in eccentric exercise as a decrease in force production at a given elongation may lead to a reduction in the level of damage to connective and muscle tissue.[53] Since muscle damage also occurs at a critical level and rate of tension during stretch, increased flexibility may also prevent stretch-induced injury. This is of particular significance for two-joint muscles that are subjected to greater levels of stretch than single-joint muscles. However, studies which have investigated the effect of stretching prior to,[7,44,53,118] after,[8,53,64,111,112,119] or before and after[120] eccentric exercise have shown no preventative effect of stretching on DOMS. One explanation is that studies that employ stretches of less than 30sec may be limited by the stretch reflex response. When a muscle is stretched the muscle spindles are also stretched, causing sensory impulses to be sent to the spinal cord to indicate that a muscle is being stretched. Efferent impulses are in turn sent back to the muscle from the spinal cord causing the muscle to contract. If the stretch continues for at least 6sec, the Golgi tendon organs send sensory impulses to the spinal cord causing a reflex relaxation of the antagonist muscle. This reflex relaxation allows the agonist muscle to stretch through relaxation, reducing the risk of damage to the muscle.[121] A short duration of stretching thus limits the time available for the Golgi tendon organs to respond to the change in length and tension of the muscles.[121] Interestingly, muscle soreness can also be induced by stretching exercises alone. Smith et al.[118] reported that three sets of 17 60-second stretches during 90 minutes of static and ballistic stretching induced significant increases in muscle soreness perception and serum CK in 20 male participants. Static stretching produced significantly greater soreness on a 10-point scale (2.1 ± 1.4) than ballistic stretching (1.6 ± 1.0) throughout the 120 hours post-exercise. Various muscles have been testing including quadriceps,[7,8,64,119,120] hamstrings,[44,53,111] calves[64] and elbow flexors[113] using stepping,[7,8,64] isokinetic profiles,[44,53,120] elbow contractions[113] and squats.[119] Further research should be directed at investigating the efficacy of stretching in order to determine its role as a preventative measure against, or a predisposing factor towards, DOMS. It is also important to emphasise that viscoelasticity is temperature-dependent.[30] Therefore, the intensity and duration of warm-up should be individualised to the athlete's physical capabilities.[121] An increase in rectal temperature of at least 1–2°C is sufficient during a warm-up, although the more practical observation of a mild sweating in normal ambient conditions has also been relia-

ble.[26,121] Despite this, only minimal warm-ups (0–5 minutes) have been reported in stretching studies.

5.3 Anti-Inflammatory Drugs

It has been proposed that the inflammatory process and the accumulation of muscle oedema following tissue injury can contribute significantly to the development of DOMS.[22,45,84] As a result, the efficacy of a number of nonsteroidal anti-inflammatory drugs (NSAIDs) and oral analgesics in preventing or treating DOMS has been examined.[17,22,37,46,62,63,122] NSAIDs inhibit the metabolism of arachidonic acid via the cyclo-oxygenase pathway and thus prevent the production of endoperoxides and prostaglandins.[62,63] A reduction in the inflammatory response leads to a reduction in the amount of muscle oedema and intramuscular pressure; two factors which contribute to pain and muscle soreness. Hasson et al.[62,63] reported significant reductions in the perception of muscle soreness at 48 hours post-exercise for an experimental group that received therapeutic or prophylactic administration of ibuprofen, dexamethasone and aspirin compared with a placebo group that received iontophoresis, or a control group who received no treatment. Maximum voluntary contraction, peak torque and work were no different between the three groups at 48 hours. Fifty percent less change in elbow extension at both 24 hours (-14 ± 3 vs $-8 \pm 1°$) and 48 hours (-30 ± 6 vs $-14 \pm 3°$) has been reported in an aspirin group when compared with a placebo group after isokinetic elbow flexor action till exhaustion at 60°/sec.[22] Soreness of the aspirin group was 25% less than the control at 48 hours. Both groups demonstrated reduced force production at 24 and 48 hours. However, other researchers[17,37,46,123] have reported no effect on the perception of muscle soreness following NSAID administration.

The inconsistency in findings may be attributed to the timing of drug administration[62] and/or the drug dosage administered.[93] Medication is generally administered when an individual complains of pain, not before. It has therefore been suggested that the consumption of an anti-inflammatory compound prior to an exercise event may alter these findings.[62] To test this hypothesis, the effect of prophylactic (prior to exercise) and therapeutic (post-exercise) drug administration has been examined. Prophylactic administration of ibuprofen (400mg three times a day for a total of 1200mg) resulted in greater declines in muscle soreness than therapeutic administration.[62] Francis and Hoobler[22] also demonstrated a reduction in muscle soreness when 10g of aspirin were administered four times daily from 4 hours prior to exercise until 48 hours post-exercise. When larger doses of NSAIDs have been administered the effect has been minimal.[37] Donnelly et al.[37] administered 1200mg of ibuprofen to 16 participants before 45 minutes of downhill running at a speed that elicited 70% heart rate maximum, and then 600mg every 6 hours up to 72 hours post-exercise. The total dose administered was 8400mg, i.e. 7-fold the dosage prescribed by Hasson et al.[62] However, despite the prophylactic and therapeutic administration in this study, the effect of the NSAIDs was minimal compared with the placebo group of 16 participants. Ibuprofen did not affect muscle soreness, muscle strength or 50% endurance time. Serum CK and urea were higher in the ibuprofen group after both runs. Large doses can impede the production of myofibrillar protein and delay the healing process of damaged tissue.[93] This was observed in a study investigating the effect of dexamethasone iontophoresis,[63] a method which involves the introduction of charged compounds across the skin and into the tissue via an electrical current (refer to section 5.4). The use of iontophoresis to administer NSAIDs resulted in a prolonged progression of muscle soreness between 24 and 48 hours.

Diclofenac does not influence muscle damage, but may reduce soreness.[124] Twenty untrained male participants took diclofenac or placebo before, and for 72 hours after, two 45 minute downhill runs 10 weeks apart. Diclofenac had no influence on the serum biochemical response to downhill running. Overall, soreness was not affected by the drug, but individual soreness was reduced by diclofenac for the first period of study. Gulick et al.[93] reported no significant differences in DOMS symptoms between

treatments (NSAID total dosage of 5400mg over 72 hours, high velocity concentric exercise, ice massage, 10 minutes stretching, arnica montana™[1] ointment and arnica montana™ pellets) for 70 untrained participants who completed 15 sets of 15 isokinetic eccentric contractions of the forearm extensor muscles. The NSAID and arnica montana™ appeared to impede recovery of muscle function. Flurbiprofen has been shown to have no effect on muscle soreness 48 hours post-exercise for six male cycle trained participants who completed three trials of 30 minutes at 80% maximal oxygen uptake on a cycle ergometer. Flurbiprofen or placebo was administered from the day before exercise until 4 days following exercise in a double-blind crossover protocol. No effect of the drug on enzyme activity was shown.[123] As a result of these findings, future research should be directed towards the use of NSAIDS as prophylactic or therapeutic interventions for post-exercise pain and discomfort. However, the encouragement of drug-use for such purposes can easily cross over to the casual abuse of other performance-related drugs, i.e. ergogenic aids and performance enhancers. The chronic overuse of NSAIDs has also been related to certain adverse effects, i.e. an increased incidence of stomach ulcers, kidney failure and liver damage.[125] Researchers must therefore recognise the potential consequences of promoting NSAID use to the general public in terms of the contraindications to anti-inflammatory drug use, and must acknowledge their responsibility as educators against drug-abuse.

5.4 Ultrasound

Ultrasound is thought to promote the inflammatory response via an increase in tissue heating and blood flow. Its success as a treatment regimen has shown mixed results. Hasson et al.[126] compared sham and real ultrasound treatment compared with a control group following 10 minutes of stepping exercise. Ultrasound given at 24 hours post-exercise to the vastus lateralis and vastus medialis and pulsed at a frequency of 1 MHz and an intensity of 0.8 W/cm^2 showed a significant reduction in soreness after 48 hours compared with the sham and control groups. In contrast, Ciccone et al.[127] reported an enhancement of DOMS symptoms following the application of ultrasound (1 MHz frequency, 1.5 W/cm^2 intensity) in 40 females following bilateral DOMS-inducing eccentric exercise of the elbow flexors. The purpose of this study was to determine the effects of ultrasound (sham and real) and/or phonophoresis using an anti-inflammatory analgesic cream (placebo or cream) during DOMS. Phonophoresis is a method of drug administration that utilises ultrasound waves to enhance the delivery of pharmacological agents through the skin to the underlying tissues. The results showed that although all groups experienced an increase in soreness 24 hours following DOMS-inducement, the group who was treated with ultrasound alone experienced the only statistically significant increase in muscle soreness at 48 hours when compared with the control arm. These increases were not observed in the group that was treated with ultrasound and trolamine salicylate cream. Therefore salicylate phonophoresis may be indicated when ultrasound is required to improve blood flow and tissue heating without promoting the inflammatory response in musculoskeletal injuries.[127] Pulsed ultrasound has also failed to diminish DOMS symptoms whether delivered once or twice daily.[128]

5.5 Electrical Current Techniques

The application of small electrical currents have been used clinically to accelerate the healing of wounds and fractures.[113] However, the efficacy of this type of treatment technique on musculoskeletal injuries is less known as only a few studies have investigated the effect of microcurrent,[129] high-volt pulsed current electrical stimulation[130] or transcutaneous electrical nerve stimulation (TENS)[131] on DOMS.

Weber et al.[87] compared the effectiveness of microcurrent electrical stimulation (30μA, gentle wave slope, 0.3Hz frequency, alternating polarity, 8

1 Use of tradenames is for product identification purposes only and does not imply endorsement.

minutes duration) in minimising muscle soreness and force deficits immediately following and 24 hours after DOMS-inducing exercise of the elbow extensors. The results showed no statistically significant difference between the microcurrent electrical stimulation group and the control group. The effect of low-volt microamperage stimulation (100μA, 0.3 pulses/sec, alternating polarity for 20 minutes) has also been explored following DOMS inducement.[113,132] When low-volt microamperage stimulation is combined with static stretching, and compared with a placebo treatment/static stretching group, no significant differences in concentric torque, pain and range of motion between groups at 24, 48, 72, 96 and 196 hours post-exercise were reported.[132] However, a transient analgesic effect was noted at 24 and 48 hours immediately post-treatment in the low-volt microamperage stimulation group compared with the placebo group. In contrast, an earlier investigation by the same authors[133] using TENS (low frequency, 2 pulses/sec, 300 μsec pulse width, 30 minutes duration) showed a significant reduction in pain perception and an improvement in range of elbow extension in eight female participants 48 hours following DOMS-inducing exercise. It has been proposed that TENS with low frequency and long pulse duration can result in the release of β-endorphin from the anterior pituitary gland. β-Endorphin shares the pre-cursor hormone propiomelanocortin with corticotropin.[134] The latter results in the synthesis and release of cortisol from the adrenal cortex which stimulates gluconeogenesis, promotes glucose utilisation, protein synthesis, fatty acid mobilisation and suppresses acute and chronic inflammatory responses.[131,135] Despite having a positive effect on pain perception and range of motion, TENS treatment showed no significant increase in serum cortisol concentration. Similar results were obtained in a study using interferential current as the intervention (low beat frequency 10 beats per second [bps] for 30 minutes vs high beat frequency 100 bps for 30 minutes).[134] Interferential current uses medium frequencies which encounter less resistance at the skin and have a better conductance through skin.[134,136] Despite these differences, the role of β-endorphin in TENS and interferential current induced analgesia remains questionable. The authors proposed that continued use of this therapeutic modality requires a more comprehensive understanding and that further investigation should include a control group to eliminate the placebo effect.[134] A recent double-blind, placebo-controlled study has shown that Acustat electro-membrane microcurrent therapy can reduce the signs and symptoms of DOMS after an eccentric exercise protocol for the arm compared with a matching placebo membrane group.[137]

5.6 Homeopathy

Homeopathy has been described as a form of therapy based on the principle of 'let like be cured with like', that is 'practitioners prescribe in a low dose whatever drug would cause symptoms similar to those in the patient were that drug to be taken in a high dose'.[16] The most popular homeopathic medicine of choice is arnica due to its analgesic, antibiotic and anti-inflammatory properties.[93,138] Recent studies,[16,93] which have investigated the effect of arnica on DOMS, have shown minimal benefit of the remedy. In a randomised, double-blind, placebo-controlled trial, no statistical differences were found in mean muscle soreness following DOMS-inducing bench stepping exercise between the placebo group and the arnica group (arnica 30c, Rhustox 30c and sarcolactic acid 30c).[16] Gulick et al.[93] reported similar results in their participants who were instructed to take three pellets (50g) of arnica montana™ in a sublingual form every 8 hours for 3 days following DOMS-inducement in the forearm extensors. When arnica was administered in an ointment format, (4%, 0.5g dose, rubbed into the skin every 8 hours for 3 days) the results were again ineffective in reducing the amount of muscle soreness when compared with a control group.

5.7 Massage

The influx of calcium ions into the muscle fibres and a subsequent disruption of the calcium homeostasis following eccentric exercise[2,81] may be restored by increasing the amount of oxygenated

blood flow to the injured area.[2] It has been suggested that an increased blood flow during vigourous massage hinders the margination of neutrophils[139] and reduces subsequent prostaglandin production, thus reducing any further damage associated with the inflammatory process. An increased delivery of oxygen also restores the mitochondrial regeneration of ATP and the active transport of calcium back into the sarcoplasmic reticulum.[2] However, studies that have examined the effect of massage on local blood flow have shown varying results. An increase in blood flow through the vascular bed during massage was reported by some researchers.[140,141] Conversely Tiidus[142] reported no differences in arterial or venous blood flow during effleurage (stroking) massage of the quadriceps. Studies examining the effect of massage on the perception of DOMS also vary. No differences in soreness levels[14,87] or force deficits[87] have been reported between the massaged limb or the control limb using either petrissage (kneading),[14] or a combination of effleurage and petrissage massage (2 minutes effleurage, 5 minutes petrissage and 1 minute effleurage)[87] following high intensity exercise.

In contrast, Rodenburg et al.[20] observed small reductions in muscle soreness following a combination of eccentric exercise of the forearm, warm-up, stretching and massage (6 minutes of skin and muscle effleurage, half a minute of tapotement/tapping, 5 minutes of petrissage and 1 minute of muscle effleurage with decreasing intensity). However, the effects of massage in this study could not be isolated from the effect of warm-up and stretching. Nonetheless, a reduction in serum CK levels in participants who were massaged (effleurage, petrissage and shaking of the limb) for 30 minutes 2 hours following exercise have been reported.[139] Similar results have been shown by Lin[119] who reported a reduction in plasma CK level after exercise when the participants in the massage group received the first 15-minute massage 2 hours after the exercise. This may suggest that the timing of massage intervention may be an influential factor. However, this remains equivocal as few studies have investigated the effects of early massage intervention (i.e. within 1–2 hours post-exercise).[21,142] Gulick et al.[93] reported that massage was not effective in abating the signs and symptoms of DOMS. The inconsistency in research findings could be attributed to the large variety of massage techniques and massage therapists.[143] The optimal duration of massage treatment also needs to be investigated as research to date has only examined the effects of 5–30 minutes of massage treatment with varied findings. It is also necessary to determine whether manual manipulation of injured tissue serves to encourage healing or impede it. The use of massage before exercise to prevent DOMS has also not been examined.

5.8 Compression

Kraemer et al.[144] reported that continuous compression was an effective therapeutic intervention in treating eccentric exercise-induced muscle soreness. Fifteen healthy, non-strength-trained men were randomly placed in a control group or a continuous compression-sleeve group. Two sets of 50 arm curls at 1 repetition maximum (RM) elbow flexion at 60 degrees/sec were completed. The compression-sleeve prevented loss of elbow extension, decreased the participants' perception of soreness, reduced swelling and promoted recovery of force production. Further studies are required to confirm the initial benefits of compression on reducing DOMS symptoms.

5.9 Hyperbaric Oxygen Therapy

Several authors have investigated the effects of hyperbaric oxygen therapy (HBOT) to enhance recovery from DOMS.[145-148] Harrison et al.[145] examined the role of HBOT in the treatment of exercise-induced muscle injury. Twenty-one college-aged males were assigned to three groups: control, 2 hours or 24 hours post-exercise. All participants performed six sets (ten repetitions per set) of eccentric repetitions with a load equivalent to 120% of their concentric maximum. HBOT treatments consisted of 100 minutes exposure to 2.5 atm and 100% oxygen with intermittent breathing of ambient air (30 minutes at 100% O_2, 5 minutes at 20.93% O_2). HBOT was not effective in the treatment of exer-

cise-induced muscle injury as indicated by isometric strength, forearm flexor cross-sectional area, serum CK levels, T2 relaxation time (via magnetic resonance imaging) and rating of perceived soreness. Mekjavic et al.[147] induced DOMS to the biceps brachii and brachialis muscles of 24 healthy male participants. Twelve participants received HBOT at 2.5 atm once a day for 7 days. The placebo group was exposed to a normoxic gas mixture at 2.5 atm. There was no difference in perceived muscle soreness or arm circumference, suggesting that HBOT therapy did not enhance recovery from DOMS. In contrast to Mekjavic et al.,[147] Staples et al.[148] suggested that HBOT may enhance recovery of quadriceps DOMS. Sixty-six untrained males had 100% oxygen at 2 atm for 1 hour per day (HBOT) or 21% oxygen at 1.2 atm for 1 hour per day (placebo). In phase one (four groups) there were no significant differences found in pain-scores between the groups over time, but the eccentric torque from immediately after exercise to 96 hours after exercise was significantly ($p = 0.021$) better in the HBOT group than in the other three groups. In phase two (three groups) there was no difference in pain-scores between the groups, but when comparing the mean torque for the sham and the 5-day HBOT groups, a significant difference ($p = 0.023$) was detected at the final test of eccentric quadriceps torque. There were no other significant differences noted between individual groups. Over-oxygenation would have resulted in oxygen toxicity, and under-oxygenation would have resulted in a lack of effect on the tissue. It was questioned whether the high-force eccentric workout was enough to induce sufficient oedema to promote tissue hypoxia. The investigators did not use biochemical or radiological markers to test the changes and only used measurement of the arm circumference for testing oedema.

5.10 Exercise

Exercise is one of the most effective strategies for alleviating DOMS.[2] However, pain relief is also temporary and rapidly resumes again following exercise cessation.[47] It has been proposed that the temporary alleviation of pain during exercise may be due to the break up of adhesions in the sore muscles, an increased removal of noxious waste products via an increased blood flow or an increased endorphin release during activity.[73] The latter results in an analgesic effect that minimises the sensation of DOMS. Elevated afferent input from large, low threshold sensory units (groups Ia, Ib and II fibres) may also interfere with the pain sensation carried by group III and IV fibres, thus reducing pain.[149]

Studies that have investigated the therapeutic effects of exercise on the development of DOMS have shown mixed findings. Upper arm ergometry performed for 8[87]–10 minutes[93] immediately following DOMS-inducing eccentric muscle activity of the elbow[87] and wrist extensors[93] revealed no statistically significant differences in muscle soreness at 24, 48 and/or 72 hours post-exercise when compared with a control group. The performance of 25 submaximal eccentric contractions 1 day following a heavy eccentric DOMS-inducing exercise regimen for the forearm flexor and extensor muscles also showed no effect on muscle soreness.[150] Hasson et al.[151] reported a significant decrease in DOMS at 48 hours following high velocity concentric isokinetic exercise (6×20 maximum voluntary contraction of the knee flexors and extensors at 5.23 rad/sec) performed 24 hours following stepping exercise. The contrast in research findings was attributed to differences in exercise protocols, including type of exercise performed, timing of exercise and degree of effort (submaximal vs maximal).[93] Therefore studies using similar exercise parameters need to be studied and compared in order to determine the true effect of exercise on reducing the magnitude and severity of muscle soreness. Research aimed at investigating the training effect of a single bout of eccentric exercise (downhill running) has shown significantly diminished muscle soreness and serum CK activity after exercise sessions repeated 3 and 6 weeks after the initial bout.[40]

It has been proposed that a continuous pool of stress-susceptible or fragile fibres are damaged by the initial bout of exercise and that this damage manifests itself as muscle soreness, enzyme changes

and a temporary reduction in the pool of fragile fibres.[40] Therefore, subsequent work bouts result in fewer stress-susceptible fibres being damaged and hence reduced muscle soreness. However, due to the dynamic degeneration-regeneration process that occurs to replace damaged fibres, the reduction in muscle soreness and serum CK levels is also temporary and exercise bouts performed at 9 weeks produced similar levels as the initial bout of exercise. This may have important implications for athletes who train on a daily basis or who are preparing for an event that will comprise some eccentric activity. In particular, attention must be paid to the scheduling of training programmes in order to minimise the amount of muscle soreness experienced at or near competition times. Future research should be directed towards the identification of pain relief strategies during exercise, and whether compensatory mechanisms are adopted to help alleviate pain. Although this may not be relevant to the recreational athlete who will most likely rest for 2–3 days following strenuous exercise, the elite athlete, who must train daily or twice daily, may be predisposed to further injury if biomechanical adaptations are adopted to help relieve intense muscle soreness.

6. Conclusions and Recommendations

DOMS can result from strenuous, unaccustomed tasks of an eccentric nature. Symptoms of DOMS can include: tenderness or stiffness to palpation (particularly at the musculotendinous junction), a loss of range of motion, flexibility, force production and mobility. Greater perception of muscle soreness tends to be associated with high intensity exercise, although duration also has a contributing effect. The impact of DOMS on athletic performance has not been well researched. However, a reduction in joint range of motion during periods of severe muscle soreness, and a reduction in shock attenuation have been observed. Significant reductions in peak torque can occur for up to 8 days following eccentric exercise, and there are also alterations in muscle sequencing and recruitment patterns which occur as a result of damage to the muscle fibres. Any compensatory mechanisms that occur may lead to an increased risk of injury to unaccustomed tissues if an individual returns to sport prematurely.

Numerous theories of DOMS have been proposed in the literature, with most criticism directed at the lactic acid theory and muscle spasm theory. It is likely that a combination of theories can contribute to DOMS with most emphasis being placed on the inflammatory response to connective tissue or muscle damage. Treatment mechanisms are also plentiful, however only limited success has been reported in research to date. The most notable findings are those related to stretching; although stretching is publicly recommended as an injury prevention measure, the rationale for stretching has yet to be validated by future research.

Future research should focus on clarifying the following points:

- Is the combination of models a valid explanation for the mechanism of DOMS? The model draws numerous aspects from different theories, therefore to assume that all these stages occur requires further investigation. The time frame of specific events also needs to be clarified.
- Is DOMS preventable prior to exercise? Are therapeutic treatment strategies suitable as prophylactic strategies? The administration of anti-inflammatory drugs, stretching and physical training are currently performed prior to exercise and have resulted in mixed findings. Can other forms of treatment also be used prophylactically to produce positive findings?
- Is stretching a valid preventative measure? If static stretching can lead to DOMS in the absence of any exercise, should individuals perform stretching at all? The increasing interest in proprioceptive neuromuscular facilitation stretching warrants investigation.
- Past research has led to the assumption that DOMS induces temporary alterations in joint kinematics and muscle activation patterns. If so, when is the safest time to return to sport and what are the biomechanical implications, if any, of premature return?

There are still many questions that remain unanswered relating to DOMS, therefore numerous re-

search directions can be pursued. This review has identified certain areas of research that may benefit both the elite and recreational athlete. However, it is important to note that DOMS is not exclusive to sport; it also has a prevalent occurrence in strenuous, unaccustomed activities of a non-sporting nature, i.e. activities of daily living. As a result, sports-related DOMS research may serve to benefit a wider audience than initially anticipated, with research findings also applicable in other health industries.

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