Mechanical Contributions to Muscle Injury: Implications for Athletic Injury Risk Mitigation

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April 9, 2020

Abstract

Muscular injury continues to be a significant burden on athletes. Through developing proper understandings of the mechanical contributions to muscle injury, appropriate intervention protocols can be established to reduce injury risk. The strength, stiffness, length, and elastic properties of the musculotendinous unit underpin the resilience of the structure. The capacity of individual muscles and tendons to absorb elastic energy, protect against high forces, resist length changes and tolerate strain encapsulate the capacity of the musculotendinous unit to avoid injury. Further, the contributions of intersarcomere dynamics to muscle injury are of utmost importance. Considering that muscle injury appears to be more strongly related to muscle fiber strain, opposed to stress, athletes should seek strategies that provide them with the capacity to resist excess strain, which is achieved through correct muscle activation timing, heightened strength and superior stiffness regulation capabilities. Further, athletes should strive to increase their capacity to withstand larger amounts of absolute strain, which can be achieved through increasing the length and maximal elongation capabilities of the structures subject to length changes, such as fascicles.

Keywords: Musculotendinous unit; Strain; Strength; Stiffness; Sarcomere; Fascicle length

DOI: <http://dx.doi.org/10.31236/osf.io/a5um4>

Citation: Kalkhoven, J. T. & Watsford, M. L., (2020). Mechanical Contributions to Muscle Injury; Implications for Athletic Injury Risk Mitigation. *SportRxiv*. *Doi:*10.31236/osf.io/a5um4

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All authors have read and approved this version of the manuscript for pre-print.
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Key Points

1. Considerable advances in the field of muscle mechanics have been achieved in the past two decades. Despite this progress, translation of this research into applied sports settings in the context of injury is limited.
2. The contributions of stress, strain, muscle activation timing, and intersarcomere dynamics are fundamental to muscle injury occurrence.
3. Based on the available literature, more extensive injury risk mitigation protocols centred on stiffness, strength and strain tolerance should be established.

1.0 INTRODUCTION

Muscular injuries are consistently the most prevalent time-loss injury across a multitude of sports including but not limited to soccer [1, 2], Australian football (AFL) [3-6], and tennis [7]. Considerably high muscular injury rates have also been reported in other sports [8-18]. Through developing proper understandings of the mechanisms contributing to muscle injury, appropriate intervention protocols can be established to increase muscular resilience and decrease the likelihood of acute muscle injury events. The strength, stiffness, length, and elastic properties of the musculotendinous unit (MTU) underpin the resilience of the relevant structures. Specifically, the capacity of individual muscles and tendons to absorb elastic energy, protect against high forces, resist length changes and tolerate strain encapsulate the capabilities of the MTU to resist injury.

The resilience of a muscle to injury and damage is largely dependent on its capacity to manage stress (applied load per cross sectional area), strain (length change), and more specifically, strain under stressful conditions [19-22]. To date, it appears that although stress and strain are inter-related, muscle damage is largely dependent on the strain experienced by the muscle rather than the stress imposed during muscle contraction [19-22]. Specifically, there is strong evidence to suggest that muscle injury typically occurs

when the muscle is subjected to strain during high levels of muscle activation and heightened tension [19-22]. When muscular stress increases, tetanic tension largely remains unchanged [20], demonstrating that muscular integrity, structural stiffness, and Young's modulus are largely maintained under high stresses. This also reflects that muscle can still work optimally and maintain large force outputs under highly stressful conditions. However, when subjected to strain muscular tension decreases [19-22], inevitably reducing stiffness and Young's modulus. This results in heightened exposure of the musculature to strain for a given applied force and reduces muscle force production, reflecting muscular impairment. Within the context of this paper, stiffness is defined as the extent to which an object resists deformation in response to an applied force while Young's modulus is defined as the ratio of stress to strain.

2.0 RELATIONSHIP BETWEEN STRESS, STRAIN AND MUSCLE INJURY

Exposure to physical stress and strain is an essential component of competing in sport and is largely unavoidable for almost all athletic pursuits. Some athletes, such as most power athletes, experience considerably large acute forces and physical stresses, exposing these athletes to a substantially greater risk of acute strain injury. There is much debate regarding the appropriate intervention strategies that best protect against muscular injury. Proper understandings of the mechanics of the MTU and the mechanisms that govern muscle injury are of utmost importance for facilitating the formation of strategies that best prevent mechanical failure (injury) of the tissue in question. This section specifically explores the mechanisms that contribute to muscle strain injuries.

2.1 Muscle strain injury

There is considerable ambiguity regarding definitions of muscle damage and injury. Recently, calls to clearly differentiate between muscle damage and injury have surfaced [23] with muscle strain injury defined as a rupture of individual muscle fibers that can extend

across a portion of fibers within a particular muscle (partial tear) or across the whole muscle (complete tear) [23, 24]. By contrast, muscle damage has been defined as a disruption to myofibrils within a muscle fiber, whereby the architecture of the sarcomeres becomes disorganized [23, 25]. A causal link between the two entities whereby microscopic damage precedes macroscopic injury has been suggested [26, 27], however, it is important to acknowledge that some authors do not distinguish between muscle damage and muscle strain injury and have described them as the same clinical entity [28, 29]. These conflicting approaches yield considerable challenges for interpreting existing research. Regardless of whether muscle damage and injury are part of the same entity or not, many common characteristics are shared. Considering the absence of a consensus surrounding the differences between muscle damage and muscle injury, they will largely be explored in tandem within the following text.

Considering that muscular injury and damage typically present when the muscle is strained during high muscle tensions, speculation that high tensions cause muscular injury is expected. However, the clinical research observing muscular injury does not support this doctrine [19, 20]. When assessing the stress/strain relationships of muscles, muscular damage appears to be more strongly related to the fiber strain (stretch/length change) magnitude rather than the absolute stress (force/pressure) imposed on the fiber [19, 20, 25]. However, the two are not mutually exclusive as more specifically, it appears that muscle injury and damage occurs when exposed to strain under conditions of high muscle tension [19-22, 30]. Lieber & Friden [20] demonstrated that in rabbit tibialis anterior muscles stimulated to maximal tetanic tensions but with differing maximal tensions due to stimulation timing (40% greater in the late stretch group), tetanic tension decreased by approximately the same magnitude in all muscles assessed when receiving the same deformation pattern (25% strain). The follow up tetanic tensions signified that both groups likely sustained similar amounts of muscle damage and muscular function impairments despite varying stress exposures, indicating that muscle damage induced by eccentric contractions was not directly related to the stress imposed upon it. Interestingly, in a similar experiment assessing the contractile and morphological properties of the tibialis anterior of rabbits in response to passive stretching, isometric contractions, and eccentric contractions, maximal tetanic tensions were reduced by 13%, 31%, and 69% respectively 1-hour post-treatment [30]. In this experiment both the isometric and eccentric contraction conditions involved maximal tetanic contractions through supramaximal stimulation of the

nerve and complete muscle activation. The greater muscular damage exhibited in the eccentric contraction group, relative to the isometric contraction group, conceivably resulted from the strain experienced rather than the absolute stress imposed on the muscle. Further, the muscle damage presented in the eccentric group relative to the passive stretch group suggested that considerably greater muscular damage specifically occurred when strain was experienced under high muscle tensions. Similar experiments have produced results that largely support the findings above [19, 21].

These results mirror standard spring and material deformation principles whereby increases in structural stiffness, which in the case of muscle results from heightened muscle tensions [31, 32], elevate the energy storage capacity of the muscle for a given strain and aid muscular strain resistance for a given applied load [19]. Suggestions that muscle activation timing may contribute substantially to muscle injury are warranted as greater levels of muscular activation are generally favorable for resisting strain due to greater stiffness and a heightened Young's modulus. In support, the force at which lengthening and subsequent injury occurs is significantly greater during muscular contraction than during passive muscular stretch [19]. Further, Butterfield & Herzog [33] showed that muscle activation timing greatly influenced muscle fiber strain magnitude, while eccentrically contracting muscles undergoing high levels of muscle activation absorbed considerably more mechanical energy than muscles with low level activation prior to muscle injury [19]. Accordingly, the correct muscle activation timing and maintenance of muscle tension under large external and internal stress, reflecting preserved muscular functioning and crossbridge formation, is important for reducing injury risk in many scenarios. However, similar to standard spring and material systems, the heightened tension-induced stiffness reduces the capacity of the structure to tolerate absolute strain. It follows that when comparing highly stiff and compliant mechanical structures, stiff structures typically display a reduced capacity to manage absolute length changes. This is reflected in the stiffer, active musculature as it is at an elevated risk of experiencing more severe injury and muscle damage compared to the more compliant, passive musculature when subjected to rapid length changes. This mechanical relationship explains, in part, why passive musculature has shown a superior capability to manage greater length changes than active musculature without being subjected to damage [19-21].

Of further importance to muscular injury understandings are the effect of strain rate on injury severity, absolute strain tolerance, and injury location. Brooks & Faulkner [34] demonstrated that greater

muscular elongation velocities increase the severity of muscular strain injury in the extensor digitorum longus muscles of mice, but only during large muscular strains. Importantly, during lesser strains the effect of strain rate on muscular injury was not significant, leading to conclusions that velocity may play a minor role in contraction-induced injury but its importance is negligible relative to that of strain. These findings were supportive of previous studies whereby strain velocity does not appear to influence muscular damage during smaller muscular strains [22, 35, 36]. Interestingly, during larger strains (30%), Warren et al. [37] also reported that a small percentage of variation in the force deficit was explained by velocity, however, this was at a relatively low strain velocity (25% maximal velocity). Despite this preliminary research, further examination of the effect of high strain rates during larger muscular strains is needed to deepen the understanding of the influence of strain rate on muscular damage and injury.

When considering injury location, Garrett et al. [38] and Best et al. [39] both investigated the effect of strain rate on muscle injury site. At strain rates of 1, 10, and 100cm/min, the vast majority of strain injuries in the tibialis anterior, extensor digitorum longus, and rectus femoris muscles of rabbits occurred at the distal musculotendinous junction (98.6%) while elongation speed had no effect on injury location [38]. In the gastrocnemius, injury still occurred at musculotendinous junctions but also at various junctions across the complex architecture of this muscle, however, elongation speed had no effect on injury location. Interestingly, at higher strain rates (100cm/s) muscle injury occurred at the distal muscle belly [39] while at slower strain rates (4cm/s and 40cm/s) injury occurred at the distal musculotendinous junction, mimicking the findings of Garrett et al. [38]. It appears that the effect of strain rate on the damage sustained by muscle is not well understood and further research is required to improve the understanding of the role of strain rate in muscle injury occurrence.

2.2 Role of active and passive components of the musculotendinous unit in strain management

To enable the establishment of intervention protocols that protect against severe muscle injury, a thorough understanding of sarcomere mechanics as they relate to muscle injury and damage is required. Some understanding of the damage that occurs within the contractile and cytoskeletal components of muscle fibers during eccentric-induced injury are of interest for determining the type of damage experienced within the muscle. According to Friden et al. [25], injuries to muscle fiber contractile and cytoskeletal components following eccentric exercise typically include Z-band streaming and dissolution [40-46], A-band disruption

[43, 47, 48], disintegration of the intermediate filament system and the loss of intracellular desmin [46, 49], and misalignment of the myofibrils [40-46]. Further, structural abnormalities following various types of exercise include primary or secondary sarcolemma disruption [50-52], swelling or disruption of the sarcotubular system [50], distortion of the myofibril contractile components [40-44, 47, 53, 54], cytoskeletal damage [49], and extracellular myofiber matrix abnormalities [55]. Of particular interest within the context of this paper are the structures that manage strain within the muscle fiber such as, titin, nebulin, desmin and the M-band as these influence and assist the maintenance of sarcomere and muscle fiber integrity [56-59]. However, it is worth noting that the loss of some filaments during eccentric loading, such as desmin, may also be attributed to enzymatic hydrolysis resulting from an influx of calcium due to muscle fiber strain as opposed to mechanical overload only [60].

In an attempt to explain muscle damage Morgan [61] describes a phenomenon whereby sarcomeres ‘pop’ when lengthened outside the range of crossbridge formation. During the ‘popped’ condition the passive structures of the sarcomere primarily support the tension. This ‘popping’ indicates the importance of the passive sarcomere structures to strain management as these structures hold a significant role in axial continuity of the myofilaments for the production of resting tension, stretch resistance and maintenance of Z-disk integrity [59, 62]. Further, it has been shown that damage to passive elements did not occur until passive fiber segments were stretched beyond thick and thin filament overlap [63]. Although fiber damage occurs during these conditions, the ‘popping’ of sarcomeres [61] may be a protective mechanism for active muscles exposed to rapid length changes as this ‘popping’ occurs in the weakest sarcomeres that are scattered throughout the length of the muscle fiber, and consequently reduce the stiffness and tension of the muscle fiber for a given fiber length. This reduction in muscular tension and stiffness would be expected to better accommodate length changes, potentially protecting the muscle from more substantial damage and injury.

In contrast to active musculature, passive stretch of the muscles does not typically produce ultrastructural damage or contractile changes [20]. Since muscle tension is minimal under passive conditions, the accompanying stiffness reduction appears to allow for greater length changes within the musculature. More specifically, in passive musculature, there are reduced linkages formed between crossbridges, reducing total muscle stiffness [61, 64-68]. It appears that under passive conditions strain is managed within the passive

components of the MTU such as tendons, connective tissue, and the non-active components of musculature [56-59]. The specific roles of the passive muscular filaments are currently a highly active area of investigation as researchers attempt to explain a number of occurrences that are currently unaccounted for in Huxley's crossbridge theory [69]. Of particular interest is the role of titin which has been identified as contributing substantially to muscle stiffness regulation. It has been suggested that titin behaves as an adaptable spring that increases engagement during muscle activation as a winding filament [70-72] with its stiffness being further modulated by the binding of calcium to the titin filament [73, 74], providing a possible contribution to the residual force enhancement phenomenon [70]. Further insight into the specific roles of the various passive filaments and proteins is outside the scope of this review, however, their inclusion within this paper is warranted to highlight their importance to maintaining muscle structure and functioning as well as the modulation of passive and active muscle stiffness.

It is important to note that cytoskeletal components of muscles are rarely damaged under passive conditions since healthy musculature, when inactive, appears to handle length changes beyond the working range of motion of the joints without being subject to injury [75]. As a result, it appears that injury occurs in other structures, such as joints, when the musculature is inactive or highly compliant [75-78]. Overall, it appears that muscle activation plays a significant role in mitigating the risk of injuries to the joint, bone, and cartilage. Further, injury to musculature may be relatively favorable considering the expedited recovery rates of musculature in comparison to joint, tendon and/or other connective tissue [79, 80]. Since individuals have the ability to alter the stiffness properties of their limbs through the activation of musculature and stretch reflex activity, it appears that humans and mammals actively adjust their stiffness properties through altering muscle activation in anticipation of, or response to large stresses or length changes within the musculature [81-85] which may have implications for fiber dynamics [33].

2.3 Intersarcomere dynamics

A more detailed exploration of the intersarcomere dynamics within single muscle fibers may provide important insights into the underlying mechanisms of muscle injury occurrence. The sarcomeres of single muscle fibers, and more specifically myofibrils, vary in both length [86, 87] and shortening velocity [68]. In frog muscle fibers, the sarcomeres closer to the ends of the fiber are typically shorter in length and shorten at heightened velocities relative to the sarcomeres closer to the centre [87]. These length discrepancies appear to

become further exaggerated during muscle activation [87]. Sarcomere-length non-uniformity [61, 88, 89], which proposes the non-uniform lengthening of sarcomeres across a muscle fiber during a number of various muscular conditions under stretch, is now widely accepted [61, 88, 89]. Considering differences in sarcomere lengthening characteristics exist, individual sarcomeres display varying yield point profiles. These discrepancies in sarcomere dynamics may also result in mismatches between sarcomeres in terms of the forces they sustain [25]. Variations in sarcomere loading and structural load tolerances may explain a number of occurrences regarding muscular injury. For example, these differences likely account for why certain sarcomeres are 'popped' beyond actin-myosin filament overlap before others, as well as why certain sarcomeres within a single fiber are at a greater risk of structural failure. Further, such events led the separation of non-uniformly lengthened sarcomeres into two distinct groups, with one set of sarcomeres at a specific point on the descending limb of the force length profile relying primarily on active force, and the other set at a specific length beyond actin-myosin filament overlap (popped sarcomeres) relying exclusively on passive force [89].

During isometric and concentric contractions, tissue stretching is typically considered to be minimal, limiting muscle damage and injury risk. Muscular damage and injury appear to be more common during eccentric contractions when compared to other forms of contraction [19-21]. Within the muscle fiber, variations in the mechanical characteristics of sarcomeres appear to result in weaker sarcomeres lengthening, and therefore experiencing strain during these types of contractions [61, 87, 90]. Interestingly, within a single muscle fiber some sarcomeres, or groups of sarcomeres, may behave isometrically or even concentrically whilst other, less stiff and actively weaker sarcomeres are lengthened outside their active range. In support of such a phenomenon Burton et al [90] and Lieber & Baskin [87] demonstrated that in frog muscle fibers the shorter sarcomeres at the extreme ends of the fiber commonly shorten while the central sarcomeres lengthen during fixed end tetani. Of further importance is the presence of a transition zone of sarcomeres which includes sarcomeres of intermediate lengths [87, 91]. These differences in sarcomere behaviour may provide a more thorough explanation of muscular injury occurrence, potentially even explaining why muscular injury has been reported, although rarely, during isometric and concentric contractions [92, 93]. In such a scenario, the strain experienced by the lengthening sarcomeres exposes them to damage [94] and a risk of mechanical failure. Further, such findings suggest that the transition phase between muscle contraction types may

be an area of interest that warrants further research in relation to intersarcomere behavior, distribution of load within muscles, and injury occurrence.

Interestingly, the primary site of muscle injury and damage in humans is much debated with some researchers suggesting that the majority of muscles appear to be most commonly injured near the musculotendinous junction at the both the proximal and distal regions [19, 24, 95] while others maintain that the mid-belly of a muscle is most at risk [96-100]. Considering the more central sarcomeres are more likely subjected to lengthening it is expected that these would be the sarcomeres at a greater risk of mechanical failure. However, several investigators have shown that the extensibility of terminal sarcomeres is reduced relative to sarcomeres throughout the rest of the muscle [86, 101] and when considering that the ends of muscles typically display a reduced cross-sectional area compared to the mid-belly, it is possible that the end regions may be most likely to experience severe injury. Muscular injury may alternatively be explained by Morgan's proposal that although the shortest sarcomeres are concentrated at the ends of a fiber, the structurally weakest sarcomeres may be scattered throughout most of the length [61]. Importantly, the majority of research in this area has not been conducted with human muscle tissue, instead focusing on animal tissue, emphasizing the need for additional investigation. The nature of intersarcomere relationships is complex and although a comprehensive exploration of this topic is outside the scope of this review, acknowledgement of the importance of these mechanical characteristics to injury occurrence is strongly emphasized. Further research into intersarcomere dynamics is clearly required to enhance the understanding of the topic as this may have considerable implications for muscular injury in athletes and the development of appropriate injury risk mitigation protocols.

3.0 CONSIDERATIONS FOR ATHLETIC INJURY RISK MITIGATION

It is well known that muscular injury risk is greater during high intensity athletic activities compared with low intensity activities, especially when high intensity eccentric loading is experienced [19, 20, 95, 102]. In elite sprinters, Yeung et al. [103] reported an injury rate of 0.87 per 1000 hours of exposure. In team sports where sprinting and kicking are common, muscular injury continues to be a major burden for athletes. Within the Australian Football League (AFL), muscular injury is the most prevalent type of injury resulting in loss of game time [3-6] with hamstring, groin, quadriceps and calf strains accounting for four

of the top five most common injuries [6]. Further, it has been demonstrated that the likelihood of muscular injury increases when AFL players perform greater amounts of high-speed running than that to which they were accustomed [104]. Parallel findings have been reported in men's professional soccer with muscular injury contributing 20–37% of all time-loss injuries [1]. Similar to the AFL, the four most common muscular injuries reported in soccer are strains to the hamstring (37%), adductor (23%), quadriceps (19%), and calf (13%) [1] with hamstring strains the most common in positions that require the greatest amount of sprinting [2, 105-109]. Other research across team sports such as rugby union [12] and NFL [2, 13, 14] as well as individual sports such as tennis [7], water skiing [15, 16], dancing [17], bull riding [15], and judo [18] further highlight the prevalence of muscular injury across the sporting arena. Considering the elevated muscle tensions, forces, strains and contraction velocities experienced during high intensity activities such as sprinting, it is evident that these activities subject the musculature to greater acute loading.

Although a multitude of research has indicated that exposure to high intensity activity increases the risk of muscular injury [104, 110], careful strategy formation to reduce injury risk in response to these findings is needed as reductions in high intensity exposure may have considerable costs to performance outcomes and may even increase injury risk when an athlete is required to partake in high intensity activities [111]. Indeed, both Duhig et al. [104] and Gabbett et al. [110] maintain that cautious training and activity load management is desired to achieve optimal athletic performance whilst ensuring athletes avoid injury. It is also important to emphasize the role of overload and high intensity activity exposure for inducing favourable adaptations that enhance athletic performance [112-116]. As such, athletes should seek strategies that encourage favourable physiological and mechanical adaptations that develop athlete resilience and best accustom athletic bodies to the heightened stresses and strains experienced during elite performance outputs.

There are a number of beneficial strategies that have reduced muscular injury risk through the development of muscular resilience and many of these may also enhance performance outcomes. Considering the primary mechanism of injury is muscular strain, athletes should seek to 1) implement strategies that increase their capacity to resist excessive muscle strain, which can be achieved through improved strength development and active stiffness regulation as well as correct muscle activation timing and 2) seek to increase the MTU's capacity to withstand larger amounts of absolute strain, which can be achieved through

increasing the length and maximal elongations of the structures subjected to length changes, such as fascicles. Interestingly, unlike common understandings of immutable, non-biological materials or spring systems where an increase in length results in a reduction in stiffness, biological matter does not necessarily share these same characteristics as they can adapt to specific stimuli [117-122]. Simultaneous increases in stiffness and length characteristics have been well documented in biological structures with one such example being the maturity of mammals from infants through to adulthood whereby tendinous and muscular structures simultaneously increase in stiffness and length during growth into adulthood [123-126]. Interestingly, it appears that exercise may also induce similar adaptations in response to various forms of training [119-121, 123, 127-141].

Considering strain is the primary mechanism by which muscular injury occurs, athletes should seek methods to resist excessive muscular strain. However, it is important to note that muscular strain and eccentric contractions are fundamental components of human locomotion and the utilisation of the stretch shorten cycle. For example, a number of researchers have determined that the hamstrings behave eccentrically during the late swing phase of sprinting [142-144] along with the stance phase [144], however, the latter is less certain. Further, eccentric muscle actions are an essential component of the counter movement jump [145]. As such, eccentric loading, for the most part, is a fundamental component of movement across a number of athletic activities and is largely unavoidable. With this in mind, athletes should seek to develop their capacity to more effectively regulate the amount of strain experienced in any given scenario and reduce the likelihood of experiencing excess strain, particularly under high loads. This can be achieved through the development of an individual's capabilities to modulate and elevate muscular tensions and stiffness. It is worth emphasizing that active muscle strength and active muscle stiffness are closely related and unlike most mechanical structures, the stiffness properties of musculature can be readily altered through an increase or decrease in muscle activation and active muscle tensions [83, 84]. The capability of an individual to readily adjust the mechanical properties of the body is highly advantageous as when muscle stretch is required, such as during passive stretching, individuals can actively reduce tension to accommodate muscle lengthening. However, in scenarios where large loads are applied and strain requires resistance, such as during heavy weight lifting or certain phases of sprinting, precise muscle activation timing, and greater amounts of muscular strength and stiffness are important.

4.0 METHODS TO ALTER MUSCULAR STIFFNESS, STRENGTH AND STRAIN TOLERANCE

Considering the benefits to muscle resilience and the multitude of reports linking stiffness, strength, and performance outputs [121, 145-158], it is of interest to explore methods to modify stiffness and strength properties of the body. Various forms of strength training have resulted in heightened stiffness and strength, including eccentric strength training [159, 160], isometric strength training [148, 161], and general weight training [121]. Increases in various lower body stiffness measures, including musculotendinous stiffness, leg stiffness, and tendon stiffness, along with strength have also resulted from plyometric training protocols [121, 148, 162]. These findings are in line with a multitude of studies exhibiting reductions in injury risk and improved athletic performance with these types of training and provide a mechanical explanation for why these training methods have a positive influence on injury risk mitigation and athletic performance outcomes.

In contrast, flexibility training has been reported to reduce stiffness both acutely [163] and chronically [164]. Increased range of motion (ROM) achieved with acute bouts of stretching has been attributed to changes in the length and stiffness of the affected limb MTU [165, 166]. Although the exact mechanisms responsible for chronic or plastic increases in ROM are debatable, the improvements have been primarily attributed to reductions in MTU stiffness [164, 167] as well as an increased tolerance to stretch [168] potentially due to a greater number of sarcomeres in series [169]. However, a number of researchers have concluded that stretching has no effect on injury risk mitigation [170-172] while others have illustrated that the most flexible individuals were more likely to suffer injuries than moderately flexible individuals [173, 174]. Furthermore, a substantial body of research appeared early in this century revealing that sustained static stretching could impair subsequent performance [175-182]. Considering these findings and the reductions in stiffness associated with static stretching, it appears that athletes should avoid static stretching unless their sporting pursuits require ROM's far beyond those typically experienced in every day athletic pursuits, such as with ballerinas. The reductions in stiffness and strength, along with the multitude of performance impairments associated with static stretching appear disadvantageous to athletes. However, dynamic and proprioceptive neuromuscular facilitation (PNF) stretching may still be of benefit to athletes, confirmed by research reporting performance improvements with these types of stretches [183-191].

A cautious approach when implementing these stretches is emphasized as they have also been associated with reduced stiffness [192, 193]. Regardless, dynamic and PNF stretching appear to be less detrimental to muscle function when compared to static stretching and may even enhance performance in some capacity despite stiffness reductions.

From a morphological perspective it is routinely reported that muscular cross-sectional area (CSA), muscle thickness and muscle volume are increased following resistance training [127, 132, 140]. Increases in these characteristics inevitably impact upon the force producing capabilities of the musculature [127, 128, 132]. CSA can be further defined as anatomical (ACSA) and physiological (PCSA) and since the PCSA represents the maximal number of actin-myosin crossbridges that can be activated in parallel during contraction, the maximal force-generating capacity of a given muscle is largely proportional to its total PCSA [127]. In addition, greater actin-myosin crossbridge formation consequently increases the stiffness of the musculature as a larger number of linkages occur during muscle activation, increasing the muscles' capacity to resist strain under highly stressful conditions. To support this, a number of researchers have noted that increases in muscle stiffness and strength reduce the amount of strain experienced by the muscle for a given force [127, 128, 130-132, 137, 140].

In combination with these muscle architectural alterations, increases in fascicle length following eccentrically focused training have been reported [129, 134, 137, 138, 140, 194]. These enhancements in fascicle length ranged from approximately 10% in the vastus lateralis to approximately 34% in the biceps femoris long head [137, 140]. Fascicle length increases are commonly understood to occur by developing a greater number of sarcomeres in series, with Goldspink [195] providing the first review on this topic, however, more evidence is needed to support this. Considering that larger fascicle lengths appear to greatly influence muscle function by exhibiting superior force-velocity and force-length relationships, and superior shortening velocities in comparison to fascicles of shorter lengths [139, 141, 196-203], further exploration in the context of stiffness and Young's modulus is warranted. Further, it has been suggested that heightened fascicle lengths may even contribute to passive energy inputs [141]. From a muscular injury standpoint, longer fascicles are beneficial due to an enhanced capacity to tolerate absolute strain. This improved strain tolerance is understood to result from the greater number of sarcomeres in series allowing for strain to be distributed across more sarcomeres, thus reducing the strain experienced by individual sarcomeres [61]. This has considerable consequences for sarcomere

functioning as less of the muscles working range would include the region of potential instability i.e. the descending limb of the length-tension curve of sarcomeres [61]. Highlighting the importance given to this, some authors even claiming that the addition of sarcomeres in series accounts for 85 to 100% of the repeated bout effect [204]. In addition, it has also been demonstrated that new sarcomeres are added in-series near the musculotendinous junction [169, 205, 206], which has been proposed as the primary site of muscular injury [38, 39]. This may have important implications for the loading of the sarcomeres located near the musculotendinous junction.

In the applied research assessing muscle architecture and injury, a wealth of research has presented a range of positive injury-related outcomes associated with increased fascicle lengths that have resulted from various forms of eccentric training. Considering the widespread prevalence of hamstring injuries across the sporting arena, the vast majority of injury research in this area has focused on the hamstrings. To date, the use of eccentric hamstring strength training is the most widely researched and recommended evidence-based strategy for hamstring injury risk mitigation [207] and has significantly reduced the risk of primary and secondary injuries (65%–85%) [208-210]. It has been proposed that eccentric strength training reduces hamstring injury risk through alterations to muscle architecture, specifically through the elongation of fascicles. Interestingly, when assessing fascicle lengths in response to 5-10 weeks of eccentric strength training, length increases of between 16% and 34% have been reported within the literature [137, 194, 211]. In contrast, one study did not report any change in fascicle lengths with 6 weeks of Nordic hamstring training [212] which may have been due to the relatively short muscle lengths experienced during this exercise, potentially limiting adaptation [213]. Of note, in professional soccer players displaying relatively short Biceps Femoris long head (BF_{lh}) fascicle lengths (<10.6cm), there was a fourfold greater risk of HSI than in players with relatively longer fascicles [214]. In the same study it was reported that for every 5 mm increase in fascicle length, HSI risk was reduced by 75%. In research assessing muscle architecture and athletic performance following injury, previously injured hamstrings expressed deficits of 30% in eccentric rate of force development on return to sport [215]. Interestingly, previous hamstring injury has also been associated with reduced fascicle lengths [216], however, owing to a lack of prospective studies, it is unclear whether these architectural changes are the cause or consequence of injury [216]. For a more detailed exploration of fascicle length and its relationship to performance and injury, Timmins et al.

[141] have presented the most up to date review on this topic.

Although the majority of training studies have centred around types of contraction and plyometric training, deeper exploration into the influence of other training considerations such as ROM, training velocity and exposure to strain rates in relation to muscle architecture and stiffness outcomes is needed. Timmins et al. [141] maintains that an intricate relationship between the ROM that a muscle group routinely undertakes and the subsequent adaptations following an intervention may exist, a notion further supported by Guex et al. [213]. They suggest that exposing a muscle to greater elongations and ROM's larger than those typically experienced on a daily basis, while adding resistance, may increase muscle fascicle length independent of contraction mode. To support this Timmins [141] cited the different responses between young and elderly adults to eccentric resistance training, as elderly individuals appear to exhibit greater increases in fascicle length than their younger counterparts [138, 217]. It was suggested that elderly persons have, on average, a habitually reduced ROM. As such, by increasing the working range of their fascicles beyond what they are accustomed to in their normal daily living, longer fascicles result. The same authors also proposed that training with an emphasis on ROM may explain why some resistance interventions have elicited no fascicle length adaptations in younger adults who may already be accustomed to the ranges of motion utilised within the training studies [218]. To date, only one study has investigated the effect of training velocity on muscle architecture, comparing the effect of fast (240°/s) and slow (90°/s) eccentric training [219]. However, the slow training group undertook their intervention through a reduced ROM (35° less than the fast training group) which may have impacted upon the outcomes. Considering the absence of research investigating the effect of specific training velocities on muscle architectural adaptations, research in this area is of high priority.

5.0 CONCLUSION

Muscular injury continues to be a significant burden on athletes. Through developing proper understandings of the mechanical contributions to muscle and tendinous injury, appropriate intervention protocols can be established to reduce injury risk. The stiffness, strength, length, and elastic properties of the musculotendinous unit underpin the resilience of the structures in question. Further, the capacity of individual muscles and tendons to absorb elastic energy, protect against high forces, resist length

changes and manage strain underpin the capabilities of the musculotendinous unit to resist injury under highly stressed conditions. It appears that although stress and strain are interrelated, muscle damage is largely dependent on the strain (stretch) experienced by the muscle as opposed to the stress (pressure/force), despite stress being present during muscle contraction. Protocols that develop the strength, stiffness regulation, and length characteristics of musculature best accustom athletic bodies to the stresses and strains experienced within their training protocols and competition events. To do this, athletes should seek strategies that provide them with the capacity to resist excessive strain, which is achieved through heightened strength and muscular stiffness. Correct muscle activation timing may also have a substantial role in injury occurrence and this should be examined in more detail in future research. Athletes should also seek to increase capacities to withstand larger amounts of absolute strain, which can be achieved through increasing the maximal elongation capabilities of the structures subject to length changes, such as fascicles.

6.0 PRACTICAL APPLICATIONS

- To reduce the risk of muscle injury, athletes should seek strategies that provide them with the capacity to resist strain. This is achieved through heightened strength, and superior stiffness regulation capabilities. Correct muscle activation timing may also have a substantial role in muscle injury risk mitigation.
- Athletes should also seek to increase their capacity to withstand larger amounts of absolute strain, which can be achieved through increasing the maximal elongation capabilities of the structures subject to length changes, such as fascicles.
- Plyometric and eccentric training appear to increase muscle strength and stiffness, CSA and fascicle lengths and should be undertaken to facilitate reduced injury risks.
- Static stretching negatively affects athletic performance and weakens tissue resilience due to reduced stiffness. In contrast, given their positive influence on neuromechanical function, dynamic and PNF stretching may be beneficial to athletic performance and should be prescribed ahead of static stretching.

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