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Commentary

Tendons under load: Understanding pathology and progression

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INTRODUCTION

Tendon pathology is a prevalent and complex issue that impacts individuals across various domains, from elite athletes to the general population. Tendons, which are specialized fibroelastic tissues that connect muscle to bone, play a critical role in enabling efficient movement and force transmission. Despite their crucial biomechanical function, tendons can be susceptible to injury and degeneration, leading to a wide range of musculoskeletal (MSK) disorders.[1] Tendinopathies represent one of the most common sources of MSK pain and dysfunction. These conditions can severely affect an individual's quality of life and athletic performance, often resulting in prolonged recovery periods and significant healthcare costs.[1-5]

The pathophysiology of tendon injuries has evolved in recent years, with emerging models providing a more nuanced understanding.[5] Conventionally, tendon injuries were classified based on an inflammatory response, often leading to the misapplication of treatments aimed at reducing inflammation.[1] However, newer research suggests that tendon pathologies are largely driven by changes in the extracellular matrix (ECM), with mechanical overload and inadequate tendon adaptation being primary contributors to degeneration.[5-7]

This article aimed to explore the latest insights into tendon structure, function, and injury classification, focusing on the evolving understanding of tendinopathy and its implications for clinical practice. Furthermore, we will review recent advancements in tendon rehabilitation strategies, emphasizing the importance of load management, progressive loading techniques, and the potential role of isometric loading as a therapeutic strategy. By examining the intersection of tendon pathology, rehabilitation, and pain management, this article seeks to provide a comprehensive overview of current research, with the ultimate goal of improving the long-term outcomes for individuals suffering from tendon-related disorders.

TENDONS: STRUCTURE, FUNCTION, INJURY CLASSIFICATION, AND EPIDEMIOLOGY

Tendons are specialized fibroelastic structures that serve as a critical interface between muscle and bone, enabling efficient force transmission and joint movement.[1] These structures play a

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fundamental role in MSK stability by regulating mechanical forces and optimizing locomotor efficiency. Structurally, tendons primarily consist of densely arranged type I collagen fibers, forming a triple-helix configuration contributing to their high tensile strength. These fibers are embedded within an ECM that facilitates biomechanical resilience. In addition, tendons possess a series of elastic components, allowing them to store and return mechanical energy – analogous to biological springs – thereby enhancing movement efficiency and reducing metabolic costs during repetitive loading cycles.[2]

The healing process of tendon injuries occurs through three distinct but overlapping phases: [1] the inflammatory or wound-healing phase,[3] the proliferative phase, and[4] the remodeling phase.[1] Tendon-related conditions represent a significant burden in both athletic and general populations. Achilles tendinopathy, for instance, affects approximately 50% of runners before the age of 45.[3] More broadly, tendinopathy is among the most prevalent MSK conditions encountered in clinical practice, frequently prompting individuals to seek medical intervention.[4]

TENDINOPATHY: TERMINOLOGY, ETIOLOGY, AND PATHOPHYSIOLOGY

The terminology associated with tendon pathology has evolved to reflect distinct pathological processes. The term tendonitis refers to an acute inflammatory condition characterized by cellular infiltration and the presence of pro-inflammatory mediators.[5] Bass (2012) characterizes tendonitis as "the inflammation of the tendon [resulting] from micro-tears that happen when the musculotendinous unit is acutely overloaded with a tensile force that is too heavy and too sudden."[5] In contrast, tendinosis describes a chronic degenerative state marked by collagen disorganization, increased ground substance, and neovascularization rather than an inflammatory response.[5] The broader term tendinopathy encompasses both inflammatory and degenerative changes, serving as a general descriptor of tendon pathology without specifying the underlying histopathological process. The primary contributing factor to tendinopathy is excessive mechanical loading that surpasses the tendon's adaptive capacity, commonly summarized as "too much, too soon" in terms of load and movement exposure.[6,7]

THE PROBLEM WITH TENDONITIS TREATMENT AND ITS EVOLVING UNDERSTANDING

Recent research into tendon pathology has raised significant concerns regarding the traditional approach to diagnosing and treating tendon injuries, particularly those termed tendonitis.[5-11] A key issue arises when researchers examine the so-called "inflamed" cells of affected tendons: Instead of typical signs of inflammation, they observe signs of degeneration. This discrepancy has led to the emergence of several theories regarding the nature of tendinopathy. One possibility suggests that untreated tendonitis progresses into tendinosis, with "itis" advancing into "osis" over time.[6] Another theory posits that tendonitis may not actually be an inflammatory condition at all, but rather a degenerative process from the outset, challenging long-held assumptions about its etiology.[6]

This evolving understanding of tendinopathy has profound implications for treatment strategies. Traditional treatments aimed at reducing inflammation – such as steroid injections, non-steroidal anti-inflammatory drugs (NSAIDs), and rest – have been shown to be ineffective in promoting long-term recovery. Furthermore, their use may even be counterproductive. According to research by Trojian and Amoako published in the ACSM Health and Fitness Journal (2015), although NSAIDs such as naproxen, ibuprofen, or corticosteroid injections may provide short-term relief, they do not facilitate tendon healing and may have a negative impact on long-term tendon health.[7] Studies have demonstrated that these treatments fail to address the underlying degenerative process, ultimately leading to poor long-term outcomes and persistent symptoms in affected individuals.[8,9]

A NEW MODEL FOR TENDON PATHOLOGY: THE CONTINUUM MODEL BY COOK AND PURDAM

The continuum model for tendon pathology, developed by Cook and Purdam,[11] represents a shift away from traditional views of tendon injuries as primarily inflammatory or due to collagen tearing. According to Cook and Purdam,[11] tendon pathology is driven primarily by cellular changes within the ECM rather than by inflammation or collagen damage. They hypothesize that inflammation exists but is unlikely to be the primary cause of pain or pathology in tendon injuries. In addition, they argue that healthy collagen is not prone to tearing, and it is only when the tendon becomes pathological that tearing can occur.[10,11]

Cook and Purdam's[11] model outlines three stages of tendon pathology, each representing a distinct phase of tissue response and degeneration:

Reactive tendinopathy (acute phase)

Reactive tendinopathy results from acute overload, trauma, or abnormal loading that exceeds the tendon's capacity. This causes a non-inflammatory matrix response, activating proliferative cells and increasing proteoglycans, leading to tendon thickening and some longitudinal separation. While swelling and pain occur, inflammation is not the primary

cause. The goal is to reduce stress on the tendon, promote adaptation, and prevent further damage. Proper management at this stage can support full tendon recovery.

Tendon disrepair (chronic phase)

In tendon disrepair, the tendon undergoes significant matrix breakdown, with increased proteoglycans, collagen separation, and matrix disorganization. This phase reflects an attempted healing response, but the tendon's structural integrity is compromised. Some reversibility is possible through appropriate management, including rest, controlled loading, and rehabilitation strategies. However, without intervention, the condition may progress into more severe degeneration.

Degenerative tendinopathy (chronic stage)

In the degenerative phase, cell death (apoptosis) and acellularity are prevalent, with well-described areas of cellular death and disordered matrix regions. The tendon matrix is highly disorganized, and little reversibility remains at this stage. Treatment focuses on managing the surrounding healthy tissue and mitigating symptoms, as the degenerated tendon cannot fully recover. Efforts are directed at improving function, reducing pain, and preventing further tendon degeneration.

This continuum model offers a more nuanced understanding of tendon pathology, emphasizing cellular changes and the role of the ECM in the progression of tendon injury, as opposed to the traditional view of tendonitis being driven primarily by inflammation or collagen tearing.[11]

THE IMPORTANCE OF LOAD IN TENDON REHABILITATION

Traditional models of tendon rehabilitation have often emphasized the need for rest as the primary treatment approach, operating under the assumption that tendinopathy, particularly tendonitis, is primarily caused by inflammation and torn tissue. In this framework, rest was seen as essential for reducing inflammation and promoting recovery. However, newer research challenges this view and prioritizes the application of mechanical load as a key driver of tendon recovery and adaptation. In this updated model, load is considered the primary factor in tendon progression and regression, not rest.[10] According to Nourissat *et al.* (2015), mechanical signals applied to tendons trigger molecular signaling pathways that initiate adaptive responses in tendon tissue. These adaptive responses lead to structural changes in the tendon, particularly an increase in collagen fibers, in response to increased mechanical load. Conversely, decreasing load leads to a reduction in collagen fiber density and a decline in tendon health.[12]

Merza *et al.* (2022) found that loading the Achilles tendon at higher intensities and durations (75% of maximal effort) led to a more pronounced temporary reduction in tendon volume and stiffness compared to lower intensity and duration (35% of max).[13] This effect was attributed to fluid redistribution within the tendon, which the authors suggested may enhance tenocyte mechanotransduction and cellular signaling. Such a response could support long-term tendon adaptation. Importantly, this highlights the potential role of heavy loading in modulating tendon fluid dynamics – an effect not observed with lighter loading – and its unique contribution to tendon remodeling.[13]

The key takeaway is that rest alone is insufficient for tendon recovery, and proper mechanical loading is required to stimulate fluid flow and tissue regeneration and prevent further deterioration. Clinical rationale is, of course, encouraged, as a reduction in load or movement may be required in the short term when handling such conditions.

ISOMETRIC LOADING: A POTENTIAL THERAPEUTIC STRATEGY FOR TENDON PAIN RELIEF

Isometric loading has emerged as a promising approach in tendon rehabilitation, particularly in managing tendon pain. Unlike fast loading (which involves rapid stretching and shortening of the tendon) and compression, which can be highly provocative and exacerbate pain, isometric loading offers a more controlled and less provocative method of stimulating tendon adaptation. Isometric contractions involve holding a muscle in a fixed position against resistance without joint movement, making them less likely to cause compression or tensile stress on the tendon, which is beneficial in the early stages of tendon recovery.[14]

An analgesic effect

A notable study by Rio *et al.* (2015) demonstrated that five sets of 45-s isometric holds at 70% maximal voluntary contraction resulted in a significant reduction in pain for up to 45 min following the exercise session. Furthermore, isometric exercise was found to reduce corticospinal inhibition – essentially "releasing the brake" on the nervous system – thereby allowing the tendon to function with less pain.[14] Rio *et al.* (2015) concluded that isometric exercise induces analgesia and reduces inhibition in patients with patellar tendinopathy, further supporting its use in tendon rehabilitation as a therapeutic strategy.[14] The study by Rio *et al.* (2015) has several limitations.[14] The small sample size limits the generalizability of the results, despite the study's cross-over design and controlled protocol. In addition, the findings are specific to patellar tendinopathy and may not apply to other types of anterior knee pain, such as patellofemoral pain, where different responses to loading may

occur. The study only included male participants, restricting the applicability of its conclusions to females. Finally, the lack of a non-intervention control group reduces the ability to assess the true effect of the intervention.

Fascicle sliding

Fascicle sliding refers to the relative movement between collagen fiber bundles within a tendon. This motion allows fascicles to glide past one another during loading, reducing internal friction, distributing mechanical stress, and enhancing the tendon's ability to adapt to strain.[15] In energy-storing tendons, fascicle sliding contributes to elasticity and efficient energy transfer during dynamic activities such as running and jumping. Its reduction after injury is linked to mechanical deficits and muscle weakness.[15] The authors (Ito *et al*., 1998) found that during isometric contractions, muscle fascicles actively shorten by approximately 15%, even though the overall muscle length remains unchanged.[16] This supports the use of isometrics as a safe and potentially analgesic approach, providing a stable environment that stimulates fascicle sliding and encourages tendon remodeling during post-injury rehabilitation.

THE IMPORTANCE OF CONTINUED LOADING FOR TENDON HEALTH

A critical study by Docking and Cook (2015) examined the structural characteristics of Achilles and patellar tendons in 91 participants using ultrasound technology (UTC).[17] To assess structural differences, the researchers compared "normal" tendons to "pathological" tendons. As anticipated, the pathological tendons were found to be thicker and exhibited more pathology than their normal counterparts. However, an important and somewhat surprising finding emerged. While pathological tendons had more structural issues, they also contained more healthy tissue compared to normal tendons. This suggests that, despite the presence of pathology, there is significant healthy tissue within the tendon that can still tolerate and benefit from loading. These findings challenge the traditional approach of excessive rest and emphasize the importance of targeted loading during rehabilitation. As Docking and Cook (2015) highlighted, we should focus on treating the "doughnut" – the healthy tissue – rather than the "hole" – the damaged area. This approach supports the idea that even in the presence of pathology, tendons can still be loaded effectively to promote adaptation and healing, especially when focusing on stimulating the healthy regions of the tendon.[17]

UNDERSTANDING THE ORIGINS OF TENDON PAIN: A COMPLEX RELATIONSHIP BETWEEN STRUCTURE, FUNCTION, AND PAIN

Tendon pain is a multifaceted and elusive phenomenon, with multiple competing theories attempting to explain its origins and mechanisms of occurrence.[18-23] One widely acknowledged observation is that sensory nerve fibers are predominantly located in the peritendon, the peripheral tissue surrounding the tendon.[21,22] This anatomical feature suggests that pain may be related to mechanical or biochemical stimulation in this region. However, several studies have demonstrated that nerve ingrowth can occur in pathological tendons, further complicating the understanding of tendon pain.[21-23] Interestingly, evidence suggests that these nerves may be sympathetic in nature, playing a role in regulating blood vessels rather than contributing directly to pain perception.[23] Other theories explore the possibility that pain could result from collagen separation and subsequent biochemical stimulation of pain pathways.[21] In addition, research on lower limb tendon pain has proposed that it is local nociceptive pain, with no central sensitization occurring, further suggesting a localized rather than systemic origin of discomfort.[20]

One crucial insight emerging from current research is that there is no direct correlation between tendon pathology and the presence of pain. In fact, pain can occur at any stage of the tendon's healing process. Interestingly, normal tendons can still be painful on imaging, while pathological tendons may not exhibit any pain at all.[18,19] This underscores the complexity of tendon pain, where structure alone does not dictate pain perception. Ultimately, the management and treatment of tendon pain should be based on a nuanced understanding of both structure and function rather than solely focusing on the imaging results or the presence of pathology.[21-23] Improving tendon function through targeted loading exercises will not only enhance tendon strength and resilience but will also lead to a downregulation of pain. In essence, the principle of function over pain should guide rehabilitation efforts, ensuring long-term recovery and minimizing reliance on short-term pain relief strategies.

TREATMENT STRATEGIES FOR TENDON PATHOLOGY: ADDRESSING FUNCTION OVER PAIN

Effective management of tendon pathology requires a multifaceted approach that targets both the nervous system and the structural function of the tendon. Treatment can be broadly classified into short-term interventions and long-term rehabilitation strategies, each addressing different aspects of the pain and healing process.[10-14,21-23]

Short-term interventions

These strategies primarily aim to modulate pain and reduce discomfort, often acting on the sensory nerves involved in the pathology. One such intervention is shock wave therapy, which has been shown to provide short-term pain relief

through its impact on the neural pathways associated with tendon pain.[9] However, other manual therapies have been used.[9] While these interventions can provide immediate relief, they do not address the root cause of the tendon dysfunction and may offer only temporary results.

Long-term rehabilitation: Structural and functional approaches

Long-term treatment must focus on the tendon's structural integrity and functional capacity. This includes exercise-based interventions, such as progressive loading strategies designed to stimulate collagen synthesis, improve tendon health, and increase the mechanical load tolerance of the tendon. Rehabilitation must incorporate energy storage, compressive forces, and friction to mimic the tendon's natural biomechanical environment, ultimately enhancing its ability to withstand daily loads and stresses.[10-14] Fast loading, as recommended in progressive rehabilitation models, is also essential for promoting tendon adaptation, encouraging collagen remodeling, and stimulating tissue healing [Figure 1].[10,11]

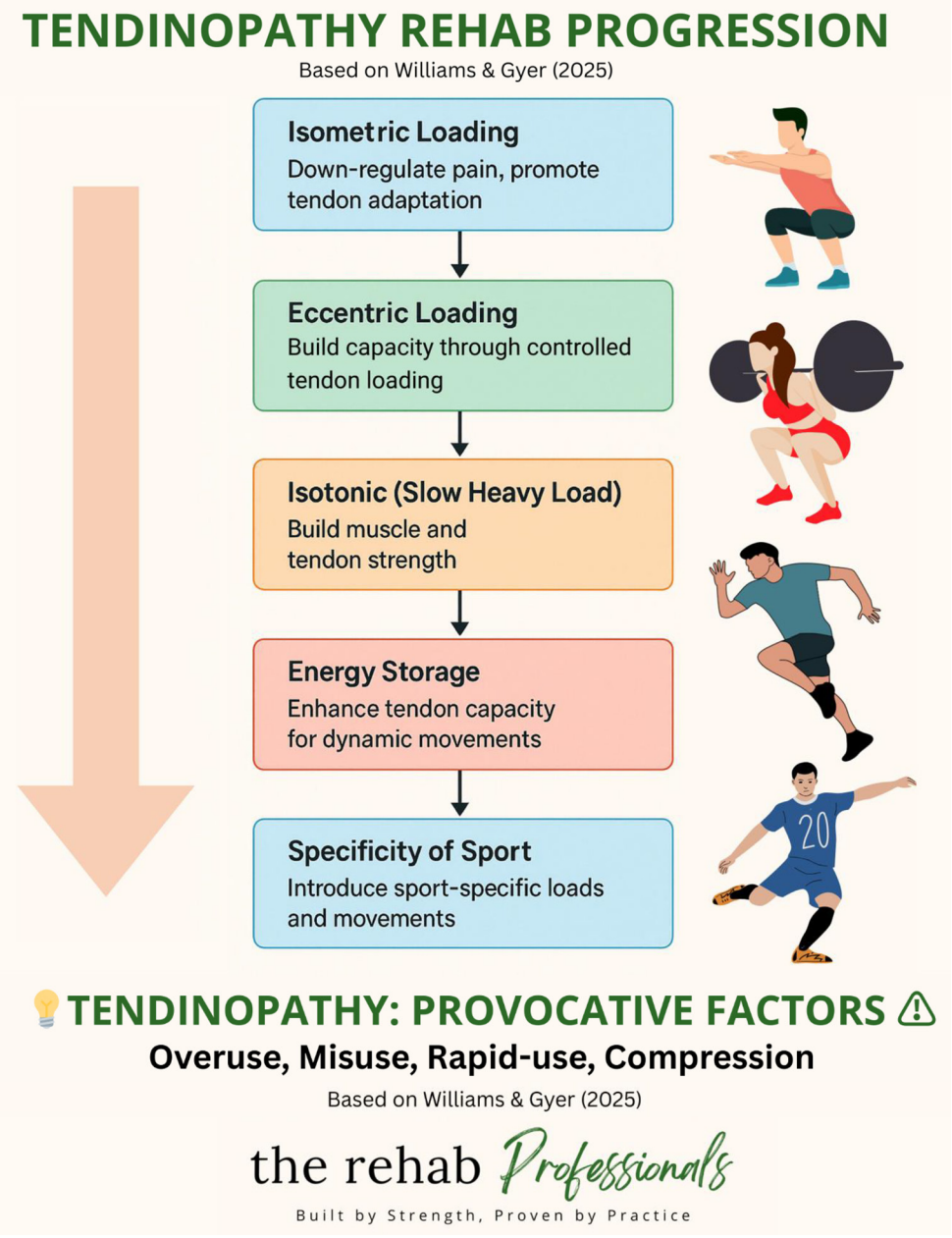

Figure 1: Rehabilitation protocols for tendon pathology. This diagram illustrates the progressive phases of tendon rehabilitation, including isometric, eccentric, isotonic slow heavy load, energy storage, and sport-specific training.

REHABILITATION PROTOCOLS FOR TENDON PATHOLOGY

Rehabilitation protocols for tendon pathology should be structured in phases that progressively load the tendon in a way that promotes healing while minimizing additional strain and provocative movements [Figure 2]. These phases can be categorized by the type of loading – isometric, eccentric, isotonic, slow heavy load, and energy storage – each addressing different aspects of tendon function and capacity [Figure 1]. However, please note, this is ultimately a guide. The right protocol should always be specific to the patient being treated. A detailed history and comprehensive assessment is always paramount.

Isometric loading

Isometric loading is an early-stage rehabilitation strategy aimed at down-regulating pain and promoting tendon adaptation without provoking further damage. This non-provocative approach avoids applying compressive stress or fast-cycle loading that could exacerbate symptoms [Figure 2]. As a result, isometric loading serves as an appropriate strategy for the early stages of tendon rehabilitation, providing pain relief and tendon activation without overloading the tendon.[14]

Eccentric loading

Eccentric loading is an effective rehabilitation tool that targets the synovio-entheseal complex at the tendon insertion site, stimulating tendon strengthening. However, eccentric loading does not address energy storage requirements (e.g., fast-cycle and rapid tendon movement) or the ability of the tendon to adapt to compressive loads. This makes eccentric loading a less provocative and early-to-mid-phase rehabilitation strategy, promoting tendon healing by improving strength at the insertion site while avoiding excessive strain [Figure 2].[10,11]

Isotonic slow heavy-load

Isotonic slow, heavy-load exercises focus on building muscle and tendon strength and are more effective in strengthening the tendon's mechanical properties. This type of loading is less effective in addressing energy storage or rapid tendon movement (fast-cycle capacity). However, it is appropriate for early to mid-phase rehabilitation, particularly regarding muscle strengthening. A structured approach should begin with isolated exercises, progress to functional movements, and then advance to kinetic chain integration and movement skill development.

Energy storage

As rehabilitation progresses, it is essential to incorporate exercises that enhance energy storage capacity, which is crucial for the tendon's ability to handle fast, dynamic movements. End-range eccentric loading and faster movements are key components of this stage, as they work to build the tendon's ability to store and release energy rapidly and restore fascicle sliding.[15] The primary goal during this phase is to increase the tendon capacity for rapid motion, preparing the tendon for more demanding and functional activities.[10,14]

Specificity of sport

As the tendon becomes more resilient and capable of handling progressive loads, it is essential to reintroduce sport-specific movements and functional loading. This ensures that the tendon is prepared for the specific mechanical demands of the athlete's sport. Loading should be tailored to the sport's requirements, focusing on loaded, powerful functional movements that mimic the dynamic nature of the sport, thereby improving performance and tendon resilience under real-world conditions.[10,11]

By gradually progressing through these stages, rehabilitation protocols aim to restore tendon function while minimizing re-injury risk.

TENDON NEUROPLASTIC TRAINING (TNT) IN TENDON REHABILITATION

Motor control deficiencies, particularly corticospinal inhibition, have been implicated in the pathophysiology of tendinopathy.[24] Even after the tendon itself has healed, these motor control deficits can persist, limiting the recovery of full functional capacity. TNT is a rehabilitation approach that specifically targets corticospinal excitability and inhibition, addressing the neurophysiological aspects of tendinopathy. This approach is often likened to "a driver who has their foot on the gas and brake at the same time," as described by Dr. Rio *et al*., illustrating the conflicting motor control signals that hinder optimal recovery.[14] The primary objective of TNT is to maximize rehabilitation and restore full tendon function. This is achieved through the use of metronome-paced strength training (MPST), which contrasts with self-paced strength training (SPST). The rationale for using MPST lies in its ability to produce greater corticospinal changes compared to SPST.[24] Leung *et al*. (2017) demonstrated that MPST evoked more significant corticospinal excitability changes than standard strength training, highlighting its potential to improve motor control and tendon rehabilitation outcomes.[25] TNT serves not only as a strategy for improving motor control but also as a means of keeping the patient engaged in their rehabilitation process. By incorporating a structured, metronome-paced approach, TNT provides an additional metric that can be used to measure progress and track the rate of loading, facilitating more precise monitoring of the patient's recovery trajectory. This TNT aspect enhances both patient

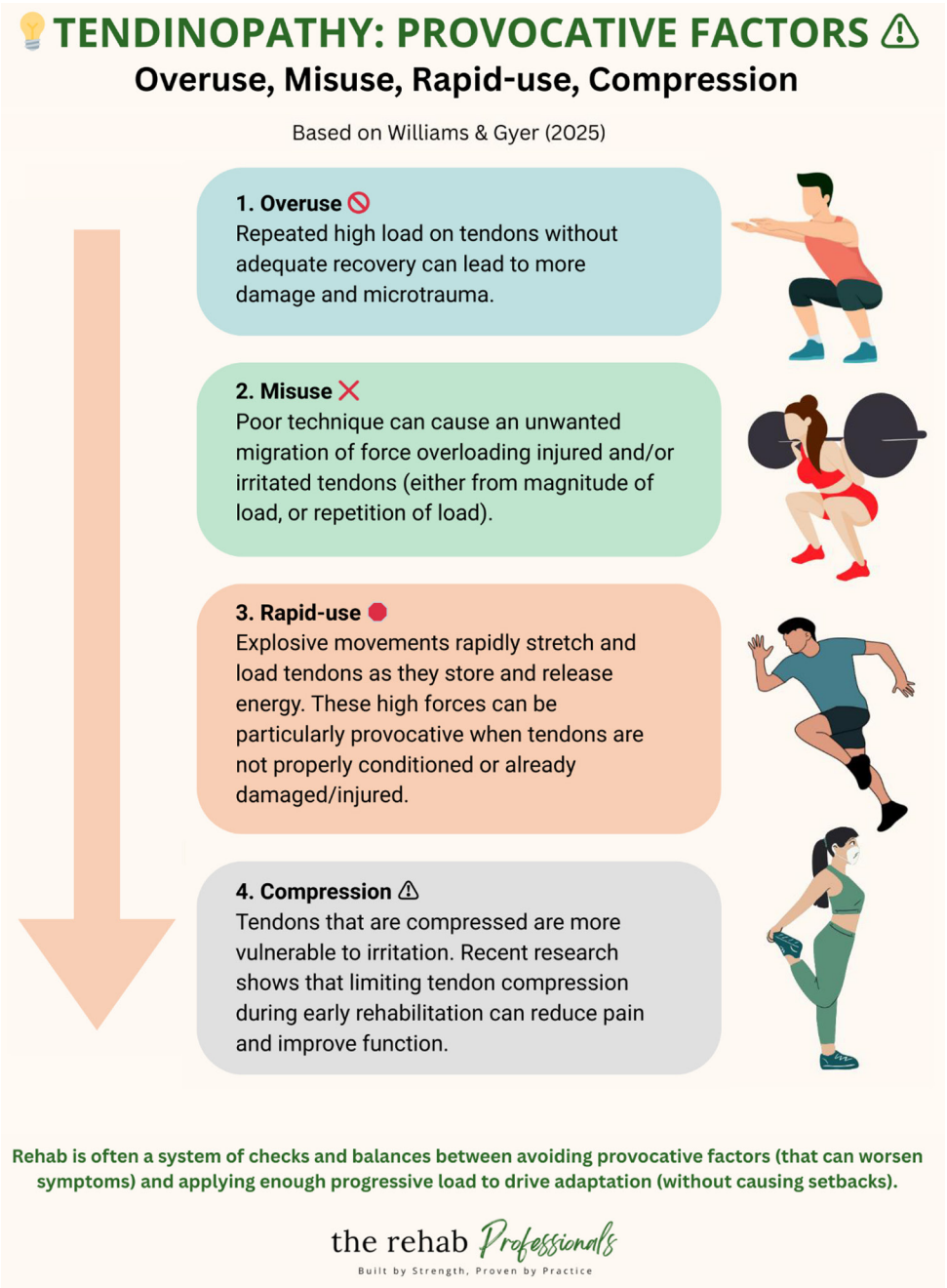

Figure 2: Tendinopathy provocative factors.

adherence and treatment efficacy, making it a valuable tool in comprehensive tendon rehabilitation.

TENDON ADAPTATION AND THE RISKS OF INADEQUATE PRESEASON TRAINING: LESSONS FROM THE 2011 NATIONAL FOOTBALL LEAGUE (NFL) LOCKOUT

Tendons require prolonged and progressive loading to achieve optimal adaptation. Due to their limited metabolic activity and restricted blood supply, tendons remodel and strengthen at a significantly slower rate than muscle tissue. When tendons are exposed to sudden, high-intensity mechanical demands without adequate preparatory loading, the risk of injury increases substantially. The 2011 NFL lockout serves as a notable case study of these risks, with a severe reduction in preseason training leading to a sharp rise in Achilles tendon ruptures.[26]

The NFL lockout, lasting 136 days from March 12 to July 25, 2011, resulted from a labor dispute between the NFL team owners and the NFL Players Association. During this period,

players were restricted from participating in organized team training, which delayed the onset of structured training programs. At the conclusion of the lockout, athletes returned to training camps with minimal time for progressive loading. Consequently, the lack of adequate preseason preparation contributed to an alarming spike in Achilles tendon ruptures, with 10 ruptures occurring within the first 12 days of camp, five of which involved rookie players. This represented a dramatic increase from the typical average of four Achilles tendon ruptures per full season.[26] This phenomenon underscores the significant risks associated with insufficient preseason training and rapid, unregulated spikes in training intensity.

The literature emphasizes that a progressive and well-structured preseason is essential for tendon health, as mechanical loading is critical to tendon adaptation and injury prevention.[27] A 24-week preseason program, compared to a 12-week or shorter alternative, allows for more gradual tendon remodeling and reduces the risk of mechanical overload. Programs incorporating eccentric loading, plyometric exercises, and controlled exposure to high-intensity movements have been shown to enhance tendon stiffness, improve neuromuscular coordination, and decrease the likelihood of tendon injury.[27] The findings from the 2011 NFL lockout provide compelling evidence for the necessity of adequate tendon preparation through a progressive, well-structured preseason training regimen. Failure to provide such preparation can lead to severe consequences for athlete health, performance, and long-term MSK health.

AGING AND TENDON HEALTH: CELLULAR, STRUCTURAL, AND FUNCTIONAL CHANGES

As individuals age, tendons undergo significant alterations at both the cellular and structural levels, which impact their functional properties, healing capacity, and overall resilience. These age-related changes present challenges for tendon repair, regeneration, and adaptation, with substantial implications for injury prevention, rehabilitation, and MSK health in older populations.[28]

Tendon stem/progenitor cells (TSPCs)

TSPCs are specialized cells responsible for tendon tissue's growth, repair, and regeneration. These cells are integral to maintaining tendon homeostasis and function. However, with advancing age, the population of TSPCs becomes progressively depleted, leading to a marked reduction in the tendon's ability to repair itself following injury. The diminished regenerative capacity of aging tendons is a key factor contributing to the prolonged healing times and decreased adaptability observed in older individuals.[28]

Cellular decline

Aging compromises tendon function through several cellular changes. The pool of TSPCs declines, reducing regenerative capacity. However, DNA levels in some tendons, like equine superficial digital flexor tendons, remain stable, suggesting DNA content alone does not explain functional decline. In addition, aging alters cell morphology, with TSPCs shifting from a spindle-like to a star-shaped form, while tenocytes become thinner and more elongated, further weakening tendon structure.[28]

Structural and mechanical changes

Aging tendons undergo structural and mechanical changes that impair function. Tendon stiffness increases due to altered TSPC properties, reducing flexibility and resilience. Cytoskeletal changes weaken tendon integrity as fibroblasts lose stress fiber density. In addition, slower actin filament dynamics hinder cell adhesion and migration, limiting the tendon's ability to respond to mechanical stimuli and repair effectively.[28]

Metabolic and functional decline

Aging reduces tendon metabolism and functional capacity. Degeneration of mitochondria and the rough endoplasmic reticulum leads to decreased protein synthesis, compromising tendon repair. Aerobic metabolism declines while anaerobic glycolysis remains stable, indicating reduced oxidative energy capacity. In addition, aged tendon fibroblasts show elevated endoplasmic reticulum stress markers, highlighting increased cellular stress and impaired homeostasis.[28]

Slower healing and regeneration

Aging significantly reduces the regenerative and healing capacity of tendons. Tendon fibroblasts divide at a much slower rate, limiting their ability to repair injuries. Gene expression shifts, with key tendon-related genes downregulated and aging-related genes upregulated, further impairing regeneration. In addition, decreased ephrin receptor activity weakens cell-cell communication, disrupting coordinated tissue repair.[28]

The aging process significantly impacts tendon health by reducing tendon tissue's regenerative capacity, flexibility, and healing potential. The depletion of TSPCs, cellular morphology alterations, and metabolic function decline contribute to the structural and mechanical changes that increase the risk of injury and delay recovery. Understanding these age-related changes is crucial for developing targeted rehabilitation strategies that account for the slower healing processes and reduced adaptability of aging tendons.

TENDON ADAPTATION AND REHABILITATION

Effective training and rehabilitation programs for older individuals must prioritize tendon health by leveraging the principles of tendon adaptation. Tendons are mechanosensitive tissues that respond to progressive mechanical loading by increasing collagen synthesis, improving fibril alignment, and enhancing stiffness – all critical for maintaining tensile strength and function. However, with age, these adaptive responses become blunted due to reduced cellular activity, mitochondrial dysfunction, and a decline in TSPCs. Despite this, research shows that tendons in older adults can still positively adapt when exposed to well-structured loading protocols. Slow, heavy resistance training has been shown to stimulate anabolic responses in aged tendons, promoting collagen turnover and improving mechanical properties. Moreover, loading at sufficient intensity and duration can mitigate degenerative changes by enhancing the metabolic activity of tenocytes and promoting tissue remodeling. These adaptations occur more slowly in older individuals, underscoring the importance of consistency, gradual progression, and adequate recovery in program design. By understanding the biology of tendon adaptation, clinicians can better tailor interventions that not only restore tendon function but also counteract the natural decline associated with aging.[12-21,28,29]

CONCLUSION

Tendinopathy remains a complex and multifaceted condition that requires a nuanced approach to both prevention and rehabilitation. The evolving understanding of tendon pathology has shifted away from an inflammatory model to one that recognizes the critical role of mechanical loading, ECM remodeling, and progressive adaptation. Research continues to highlight the importance of load management, with evidence supporting the use of structured, progressive loading to enhance tendon resilience, modulate pain, and promote tissue repair.

Furthermore, aging tendons present unique challenges, as alterations in collagen turnover, reduced cellular responsiveness, and compromised healing capacity necessitate early and individualized interventions. By integrating biomechanics, rehabilitation science, and emerging therapeutic modalities, clinicians can develop more effective treatment strategies tailored to each patient's specific needs.

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REFERENCES

1. Li ZJ, Yang QQ, Zhou YL. Basic research on tendon repair: Strategies, evaluation, and development. Front Med (Lausanne) 2021;8:664909.
2. Holt NC, Roberts TJ, Askew GN. The energetic benefits of tendon springs in running: Is the reduction of muscle work important? J Exp Biol 2014;217:4365-71.
3. Gaida JE, Alfredson H, Kiss ZS, Bass SL, Cook JL. Asymptomatic Achilles tendon pathology is associated with a central fat distribution in men and a peripheral fat distribution in women: A cross sectional study of 298 individuals. BMC Musculoskelet Disord 2010;11:41.
4. McCormick A, Charlton J, Fleming D. Assessing health needs in primary care. Morbidity study from general practice provides another source of information. BMJ 1995;310:1534.
5. Bass E. Tendinopathy: Why the difference between tendinitis and tendinosis matters. Int J Ther Massage Bodywork 2012;5:14-7.
6. Lemont H, Ammirati KM, Usen N. Plantar fasciitis: A degenerative process (fasciosis) without inflammation. J Am Podiatr Med Assoc 2003;93:234-7.
7. Trojian TH, Amoako A. Tendinopathy not tendonitis: Now is the time for a change. ACSM Health Fit J 2015;19:37-42.
8. Stahl S, Kaufman T. The efficacy of an injection of steroids for medial epicondylitis. A prospective study of sixty elbows. J Bone Joint Surg Am 1997;79:1648-52.
9. Andres BM, Murrell GA. Treatment of tendinopathy: What works, what does not, and what is on the horizon. Clin Orthop Relat Res 2008;466:1539-54.
10. Canosa-Carro L, Bravo-Aguilar M, Abuín-Porras V, Almazán-Polo J, García-Pérez-de-Sevilla G, Rodríguez-Costa I, *et al*. Current understanding of the diagnosis and management of the tendinopathy: An update from the lab to the clinical practice. Dis Mon 2022;68:101314.
11. Cook JL, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med 2008;43:409-16.
12. Nourissat G, Berenbaum F, Duprez D. Tendon injury: From biology to tendon repair. Nat Rev Rheumatol 2015;11:223-33.
13. Merza EY, Pearson SJ, Lichtwark GA, Malliaras P. The acute effects of higher versus lower load duration and intensity on morphological and mechanical properties of the healthy Achilles tendon: A randomized crossover trial. J Exp Biol 2022;225:jeb243741.

14. Rio E, Kidgell D, Purdam C, Gaida J, Moseley GL, Pearce AJ, *et al*. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. Br J Sports Med 2015;49:1277-83.
15. Thorpe CT, Godinho MS, Riley GP, Birch HL, Clegg PD, Screen HR. The interfascicular matrix enables fascicle sliding and recovery in tendon, and behaves more elastically in energy storing tendons. J Mech Behav Biomed Mater 2015;52:85-94.
16. Ito M, Kawakami Y, Ichinose Y, Fukashiro S, Fukunaga T. Nonisometric behavior of fascicles during isometric contractions of a human muscle. J Appl Physiol (1985) 1998;85:1230-5.
17. Docking SI, Cook J. Pathological tendons maintain sufficient aligned fibrillar structure on ultrasound tissue characterization (UTC). Scand J Med Sci Sports 2015;26:675-83.
18. Malliaras P, Cook J. Patellar tendons with normal imaging and pain: Change in imaging and pain status over a volleyball season. Clin J Sport Med 2006;16:388-91.
19. Cook JL, Khan KM, Kiss ZS, Purdam CR, Griffiths L. Prospective imaging study of asymptomatic patellar tendinopathy in elite junior basketball players. J Ultrasound Med 2000;19:473-9.
20. Plinsinga ML, Van Wilgen CP, Brink MS, Vuvan V, Stephenson A, Heales LJ, *et al*. Patellar and Achilles tendinopathies are predominantly peripheral pain states: A blinded case control study of somatosensory and psychological profiles. Br J Sports Med 2017;52:284-91.
21. Fredberg U, Stengaard-Pedersen K. Chronic tendinopathy tissue pathology, pain mechanisms, and etiology with a special focus on inflammation. Scand J Med Sci Sports 2008;18:3-15.
22. Witonski D, Wagrowska-Danielewicz M. Distribution of substance-P nerve fibres in the knee joint in patients with anterior knee pain syndrome. A preliminary report. Knee Surg Sports Traumatol Arthrosc 1999;7:177-83.
23. Ackermann PW, Salo P, Hart DA. Tendon innervation. Adv Exp Med Biol 2016;920:35-51.
24. Rio E, Kidgell D, Moseley GL, Gaida J, Docking S, Purdam C, *et al*. Tendon neuroplastic training: Changing the way we think about tendon rehabilitation: A narrative review. Br J Sports Med 2015;50:209-15.
25. Leung M, Rantalainen T, Teo WP, Kidgell D. The corticospinal responses of metronome-paced, but not self-paced strength training are similar to motor skill training. Eur J Appl Physiol 2017;117:2479-92.
26. Myer GD, Faigenbaum AD, Cherny CE, Heidt RS Jr., Hewett TE. Did the NFL lockout expose the Achilles heel of competitive sports? J Orthop Sports Phys Ther 2011;41:702-5.
27. Galloway MT, Lalley AL, Shearn JT. The role of mechanical loading in tendon development, maintenance, injury, and repair. J Bone Joint Surg Am 2013;95:1620-8.
28. Kwan KY, Ng KW, Rao Y, Zhu C, Qi S, Tuan RS, *et al*. Effect of aging on tendon biology, biomechanics and implications for treatment approaches. Int J Mol Sci 2023;24:15183.
29. Tedeschi R, Platano D, Donati D, Giorgi F. Functional approaches in tendinopathy rehabilitation: Exploring the role of tendon neuroplastic training. Manuelle Med 2024;62:1-7.