Recovery Strategies After Tendon Injuries in Athletes: A Review of Current Evidence

Maria Arshad[1], Dana F. Flavin[2] and Jamal Abdul Nasir[3]

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[1]Department of Biotechnology, Fatima Jinnah Women University, Rawalpindi, Pakistan
[2]Department of Human Sciences, Guglielmo Marconi University, Via Plinio, 44, 00193 Rome, Italy
[3]Department of Chemistry, Kathleen Lonsdale Materials Chemistry, University College London, London, UK

Correspondence to: Jamal Abdul Nasir, jamal.nasir.18@ucl.ac.uk

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ABSTRACT

Tendon injuries are a prevalent and challenging concern in athletic populations, often leading to prolonged downtime, impaired performance, and risk of reinjury. A diverse array of mechanisms can contribute to the development of tendinopathy or tendon rupture. This review discusses the current evidence on recovery strategies post-tendon injury, focusing on the mechanisms of tendon healing, rehabilitation protocols, and return-to-sport criteria. Emphasis is placed on the biological and mechanical aspects of tendon regeneration, alongside clinical applications such as eccentric loading, regenerative protocols, and neuromuscular re-education. Furthermore, the role of emerging modalities, including platelet-rich plasma (PRP), shockwave therapy, and nutritional supplementation, is evaluated. The review also highlights gaps in the literature and suggests directions for future research to optimise recovery and performance outcomes in athletes.

Keywords: Tendon injuries, Regenerative strategies, Platelet-rich plasma, Mesenchymal stem cells, Return-to-sport criteria

Introduction

Tendons serve as the critical anatomical link between muscle and bone, transmitting the forces generated by muscle contractions to the skeletal system to facilitate movement and support posture (Figure 1a).[1] Although structurally similar, ligaments differ by connecting bone to bone and primarily function to stabilise joints, typically enduring lower tensile loads than tendons.[10] Structurally, tendons are composed of a hierarchical arrangement of collagen, beginning with type I collagen molecules that form triple helices. These molecules aggregate into fibrils, which then organise into fibres, fascicles, and ultimately the full tendon structure (Figure 1b). The collagen fibrils, aligned along the axis of muscle pull, are embedded densely within the extracellular matrix (ECM) and represent the primary units responsible for transmitting mechanical force (Figure 1).[1]

Tendon injuries are a prevalent concern in athletes, often resulting in prolonged recovery times, performance limitations, and high reinjury rates.[2] These injuries range from acute ruptures caused by sudden overloads to chronic tendinopathies associated with repetitive strain or degenerative changes. Tendon disorders significantly contribute to pain, disability, and loss of productivity in both recreational and professional sports settings.[3]

The mechanisms underlying tendon injury are multifactorial. Acute tears may occur in otherwise healthy tendons during high-impact activities, while chronic tendinopathies often result from accumulated microtrauma or intrinsic degeneration, particularly with ageing.[4] For instance, while flexor tendon injuries typically result from laceration and are more prevalent in young working-age males,[5] Achilles tendon ruptures often represent acute events superimposed on chronically degenerated tissue, especially in men aged 30–49 engaging in recreational sports.[6] Similarly, rotator cuff pathology is highly prevalent in older individuals, with age-related degeneration and other systemic factors such as smoking and hypercholesterolemia contributing to tendon vulnerability.[7]

Healing outcomes are highly variable and depend on both the anatomical context and local environment. Intrasynovial tendons like the flexor and rotator cuff tendons exhibit limited spontaneous healing capacity due to poor vascularity and mechanical complexity, whereas extrasynovial tendons such as the Achilles can undergo fibrous tissue regeneration post-injury.[7] Despite progressing through the classical phases of inflammation, proliferation, and remodelling, tendon healing often results in scar tissue with inferior mechanical properties compared to native tendon.[8] Animal models have played a pivotal role in advancing our understanding of tendon injury and repair. Models using rodents, rabbits, sheep, and dogs have been employed to mimic both acute and chronic tendon injuries, enabling researchers to study healing processes and evaluate therapeutic interventions in a controlled manner.[9,10] These models also allow for investigation into post-injury biomechanics, biological augmentation strategies, and rehabilitation protocols relevant to athletic populations.

Given the complex biological, mechanical, and environmental factors that influence tendon healing, this review aims to critically evaluate current evidence on recovery strategies following tendon injuries in athletes. It will explore established and emerging approaches in conservative management, surgical repair, biological therapies, and functional rehabilitation, with a focus on return-to-sport outcomes. Emphasis is placed on translating scientific insights into clinically relevant practices that enhance functional recovery and reduce reinjury risk in competitive settings.

This review particularly offers a structured narrative overview that integrates current knowledge from cellular biology, regenerative strategies, clinical rehabilitation, and return-to-sport (RTS) outcomes. By drawing connections across these domains, the review is intended as a pedagogical resource for clinicians, researchers, and sports practitioners seeking to align biological mechanisms with practical recovery protocols.

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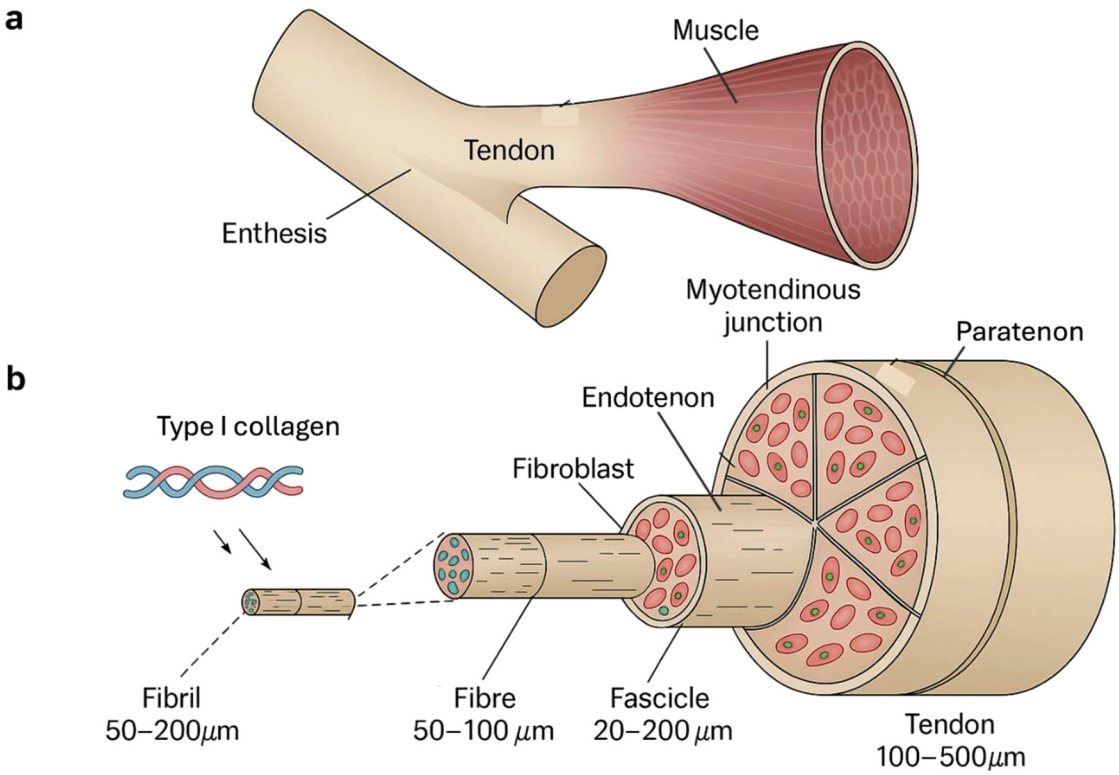

Fig 1 | a) Tendons connect muscles to bones, anchoring to bone at the enthesis and to muscle at the myotendinous junction. They are primarily made of type I collagen fibres arranged in a highly organised spatial structure. b) Type I collagen is the main structural element of tendons, which produces the α1(I) and α2(I) collagen chains. These chains form triple-helical molecules, typically composed of two α1(I) and one α2(I) chains, which assemble into fibrils that group into larger collagen fibres. Fibres are enclosed by the endotenon, a connective tissue that also houses fibroblasts. Multiple fibre bundles together to form fascicles. The entire tendon is wrapped by an outer connective tissue layer called the epitenon, which itself is surrounded by the paratenon

Methodology

A comprehensive literature search was conducted to identify studies addressing recovery strategies after tendon injuries in athletes. The following databases (illustrated in Figure 2) were systematically searched: PubMed, Scopus, Web of Science, and Google Scholar.

Search Strategy

Search terms included combinations of the following keywords: Tendon injury, tendinopathy, tendon rupture, tendon healing, athlete, sports injury, rehabilitation, physical therapy, regenerative therapy, platelet-rich plasma, stem cells, and growth factors, return to sport. Boolean operators (AND, OR) were used to combine terms. An example search string:

("tendon injury" OR "tendinopathy" OR "tendon rupture") AND ("athlete" OR "sports") AND ("rehabilitation" OR "recovery" OR "regenerative therapy" OR "PRP" OR "stem cells")

Time Frame and Language Limits

Studies published from **1980 to 2025** were considered. Only **English-language** publications were included.

Inclusion Criteria

- Original research studies, systematic reviews, and meta-analyses.
- Studies reporting recovery, rehabilitation, or regenerative interventions after tendon injuries.
- Studies conducted in **athletic populations** or relevant experimental models.

Exclusion Criteria

- Studies not focused on tendon recovery or rehabilitation.
- Case reports, editorials, or conference abstracts without full text.
- Non-English publications.

Study Selection

After removal of duplicates, titles and abstracts were screened independently by two reviewers. Full texts of potentially eligible studies were assessed for inclusion. Discrepancies were resolved by discussion.

Data Extraction

Data were extracted on study design, population, and type of tendon injury, intervention type, outcome measures, and main findings.

Study-Selection Flow

The study-selection process is summarized in the following PRISMA-lite flow diagram.

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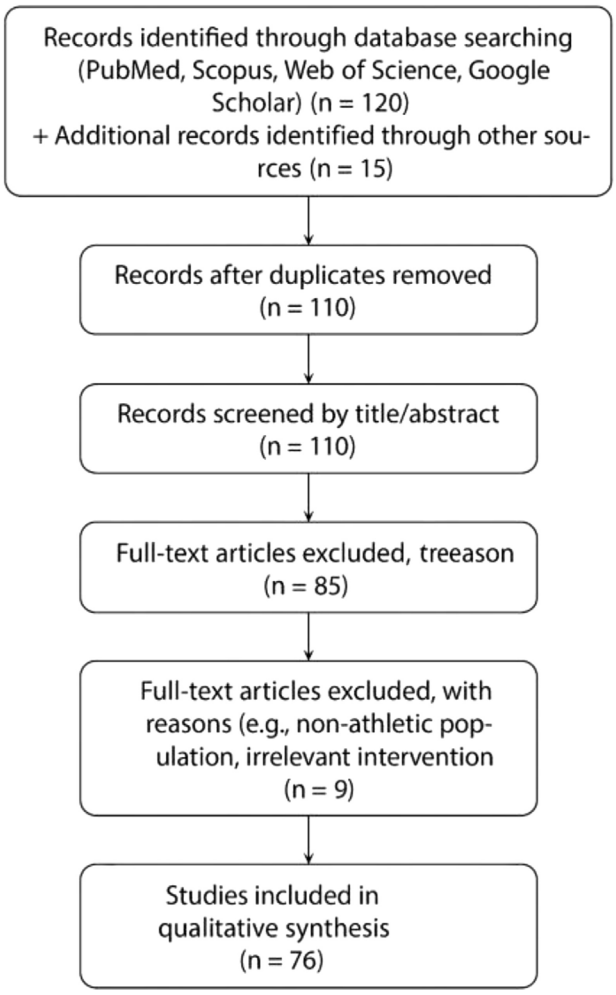

Fig 2 | PRISMA-lite flow diagram studies addressing recovery strategies after tendon injuries in athletes

Pathophysiology of Tendon Injury and Healing

Tendon injuries are a major concern in both elite and recreational athletes, presenting in a spectrum that includes acute lacerations, ruptures, and chronic tendinopathies. The pathophysiological basis of these injuries is multifactorial, involving both extrinsic overload and intrinsic tissue degeneration. Acute ruptures often occur in previously healthy tendons subjected to sudden, high-magnitude forces, such as during sprinting or jumping. Conversely, chronic tendinopathies typically develop from repetitive microtrauma, ageing, or metabolic derangements that compromise the extracellular matrix (ECM) integrity over time.

Tendon Structure and Vulnerability

Tendons are composed of dense collagenous tissue, primarily type I collagen, organised in a hierarchical structure that transmits force from muscle to bone. Their biomechanical properties depend heavily on collagen fibre orientation, ECM composition, and cellular health. Tendon vascularity is typically low, and some anatomical regions (e.g., intrasynovial tendons) are nearly avascular, which limits their capacity for spontaneous repair. These structural features contribute to their vulnerability, especially under conditions of repetitive stress or a poor regenerative environment.[7]

Distinct Tendon Environments

The anatomical location and synovial environment play a crucial role in injury susceptibility and healing outcomes. For example, intrasynovial tendons, like the flexor tendons and rotator cuff, are encased in a synovial sheath and do not heal spontaneously due to limited vascular supply and an immunologically isolated environment. Healing in these tendons is also complicated by adhesion formation, particularly in the flexor system, where scarring limits tendon gliding.[11,12] In contrast, extrasynovial tendons, such as the Achilles tendon, exhibit greater vascularity and often show a fibrotic repair response after injury. Notably, even in cases of Achilles rupture, often seen in middle-aged athletes, histological examination frequently reveals preexisting degenerative changes, highlighting that what appears to be acute trauma is often superimposed on chronic pathology.[13]

Cellular and Molecular Healing Responses

Inflammation (Days 0–7): Characterised by increased vascular permeability and infiltration of neutrophils and monocytes. These cells secrete cytokines that recruit macrophages and resident fibroblasts to the injury site. This inflammatory response sets the stage for matrix remodelling but can also promote fibrosis if unchecked.[8,14]

Proliferation (Days 7–21): Fibroblasts proliferate and secrete type III collagen, glycosaminoglycans, and other ECM proteins. Neovascularisation is prominent. However, in flexor tendons, the repair tissue may adhere to the synovial sheath, forming fibrotic adhesions that limit mobility.[12] Remodelling (Weeks to Months): Type III collagen is gradually replaced by mechanically superior type I collagen. Collagen fibres realign according to mechanical loading, and crosslinking improves tensile strength. However, full mechanical recovery is rarely achieved, especially in high-demand athletes.[15]

Role of Macrophages and Inflammation Resolution

Macrophages are central to orchestrating both injury response and resolution. Initially, M1 macrophages dominate, secreting pro-inflammatory cytokines (e.g., IL-1β, TNF-α) that initiate tissue clearance and stimulate ECM deposition. Later, M2 macrophages help resolve inflammation by producing anti-inflammatory mediators (e.g., IL-10, TGF-β1) and facilitating tissue remodelling.[16] An imbalance between these phenotypes—either sustained M1 activation or excessive M2 activity—can impair healing. M1 dominance may drive chronic inflammation and fibrosis, whereas prolonged M2 activity can lead to excessive ECM remodelling and structural weakness. This highlights the importance of timing and regulation in the inflammatory cascade.[17]

Molecular Drivers: Growth Factors and Signalling Pathways

A variety of growth factors modulate tendon healing. Platelet-derived growth factor (PDGF) and basic

fibroblast growth factor (bFGF) stimulate fibroblast proliferation and collagen synthesis. However, excessive bFGF activity may contribute to adhesion formation in flexor tendons.[18] Transforming growth factor-beta (TGF-β) is critical for ECM production and remodelling, but its isoforms play distinct roles: TGF-β1 and TGF-β2 promote scarring, while TGF-β3 is associated with regenerative, scarless healing observed in fetal tendons.[19] Although growth factor therapies hold promise, results have been inconsistent. The complexity of dosing, delivery timing, and synergistic interactions complicates therapeutic translation. For instance, attempts to modulate TGF-β isoform expression in rotator cuff models did not yield regenerative healing outcomes, emphasising the need for precise molecular control.[20]

Influence of Mechanical Loading

Mechanical loading plays a dual role in tendon healing. While early controlled loading can improve collagen alignment and tensile strength, excessive or premature loading may cause repair site gapping or rupture. Conversely, prolonged immobilisation can promote adhesions and limit functional recovery. Thus, an optimal "mechanobiological window" must be identified for each tendon type and injury context.[21,22] In rotator cuff repairs, for example, some studies suggest initial immobilisation may be beneficial to prevent early gapping, whereas flexor tendons require early passive motion to avoid adhesion formation. These biomechanical considerations are fundamental to designing effective rehabilitation protocols post-injury.[23]

Harvey et al.[24] explored the dual function of PDGFRα signalling in both tenocyte formation and tendon fibrosis. By introducing PDGF-AA (platelet-derived growth factor-AA) into the patellar tendon, they observed a rise in Tppp3$^+$ lineage-derived tenocytes, alongside an increase in ER-TR7$^+$ reticular fibroblasts. To determine the necessity of PDGFRα signalling in tendon repair, the researchers selectively deleted PDGFRα in Tppp3$^+$ cells and tracked their fate. The findings revealed that PDGFRα is essential for the expansion and differentiation of tendon stem cells into tenocytes. Moreover, injured mice lacking PDGFRα in Tppp3$^+$ cells exhibited a marked increase in ER-TR7$^+$ fibroblasts, implying that failed regeneration due to impaired TSC function allows T-FAPs to drive excessive fibrotic tissue formation (Figure 3).

Diagnosis and Classification of Tendon Injuries

The accurate diagnosis and classification of tendon injuries, particularly in the subscapularis (SSC) tendon, is crucial for appropriate treatment planning, rehabilitation, and predicting clinical outcomes in athletes. Misdiagnosis or underestimation of tendon pathology may result in persistent pain, mechanical dysfunction,

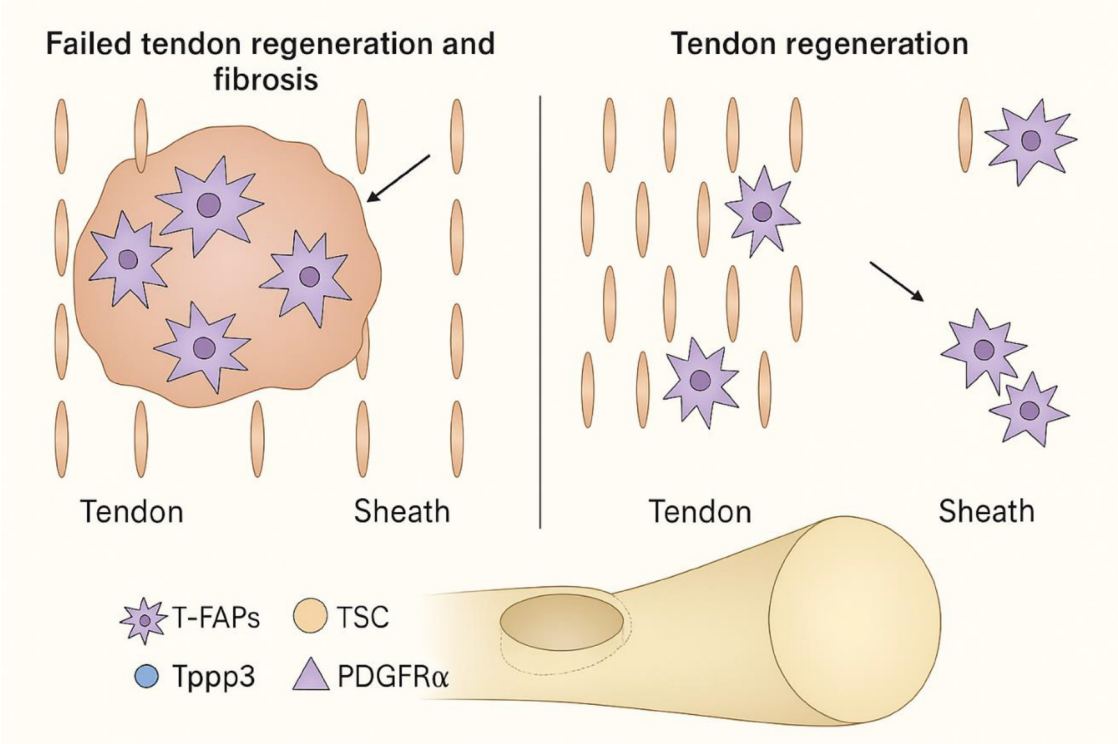

Fig 3 | Schematic of two cell populations reported by Harvey et al.[24] and their roles in tendon healing. One cell population, known as tendon stem cells (TSCs), is identified by the expression of Tppp3$^+$/pdgfra$^+$ and is involved in tendon regeneration. A second population, referred to as tendon fibro–adipogenic progenitors (T-FAPs), contributes to tendon fibrosis. PDGFRα signalling is critical for both the formation of tenocytes and the development of fibrosis. In the absence of PDGFRα signalling, TSC-driven regeneration is compromised, allowing T-FAPs to become predominant and resulting in increased fibrosis

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or failure of surgical repair, particularly in overhead or contact sports athletes who place high functional demands on the shoulder joint.[25]

Clinical assessment remains a cornerstone in the initial evaluation. Among the clinical tests employed for SSC tears, the lift-off, belly-press, and bear-hug tests are most commonly used and exhibit relatively high specificity but limited sensitivity.[26] These tests aim to detect internal rotation weakness and compensatory motion, although their diagnostic yield may vary with the chronicity and severity of the tear. In their meta-analysis, Saremi and Seifrabiei reported pooled sensitivity and specificity of 0.49 and 0.89, respectively, for clinical assessment in diagnosing SSC tendon tears.[27]

However, imaging modalities are often required to confirm the diagnosis, define the extent of injury, and assist in preoperative planning. Magnetic Resonance Arthrography (MRA) has emerged as the most sensitive imaging modality, with a pooled sensitivity of 0.83 and diagnostic accuracy of 0.85, making it the most effective tool for detecting subtle or partial-thickness tears of the SSC.[26,28] MRI, though slightly less sensitive (0.71), was shown to have the highest specificity (0.93), which suggests a strong role in confirming suspected full-thickness tears.[29]

Ultrasonography, while advantageous in terms of accessibility and cost, demonstrated limited sensitivity (0.39) for SSC tears, despite its high specificity (0.93).[30] The relatively poor sensitivity restricts its utility as a standalone diagnostic tool for subscapularis pathology. Additionally, Computed Tomography Arthrography (CTA), although evaluated in only one study, showed promising diagnostic potential with both sensitivity and specificity values at 0.90, highlighting its role as an alternative when MRI is contraindicated or unavailable.[31]

Arthroscopy, while invasive, remains the gold standard for diagnosing subscapularis tears, offering direct visualisation of tendon integrity and footprint involvement.[32] Nonetheless, its invasiveness and cost limit its use to surgical planning or ambiguous cases. Alongside diagnosis, various classification systems have been developed to characterise the extent and pattern of SSC tendon tears. The Lafosse classification, perhaps the most widely used, categorises tears from partial superior one-third lesions (Type I) to complete ruptures with tendon retraction and fatty degeneration (Type V).[33] This system provides a pragmatic grading for arthroscopic repair decision-making.

Other classification schemes offer more nuanced descriptions. Fox and Romeo classified tears by percentage of involvement (from partial to complete tears),[34] while Lyons described them in terms of thickness and retraction. The Toussaint classification emphasises the involvement of the bicipital sling alongside tendon damage, while Martetschläger et al. focused on partial tear size, measuring lesion width in millimetres.[35] The Yoo and Rhee classification is based on a three-dimensional anatomic footprint and divides tears into five types, incorporating concealed and visible lesions.[36] Finally, Dierckman's classification uniquely addresses non-insertional tears, including interstitial or degenerative variants not captured by traditional systems.[37]

Despite these numerous schemes, no universal consensus exists on a single classification model. This lack of standardisation complicates clinical comparison and treatment benchmarking across studies. Furthermore, most classification systems are based on arthroscopic or anatomic data, limiting their utility in early, imaging-only diagnoses. Notably, imaging accuracy can vary with the reviewer's expertise. The accuracy of MRI and MRA interpretation improves significantly in experienced hands and is not dependent on the time elapsed from injury to imaging, underscoring their reliability for both acute and chronic SSC tears.[38]

Regenerative and Surgical Interventions

Evidence Appraisal

The body of evidence on tendon injury recovery strategies has been appraised using a modified GRADE (Grading of Recommendations Assessment, Development and Evaluation) framework.[39,40] This approach enables transparent classification of the certainty of available data and has been adapted from previous frameworks used in musculoskeletal injury research, including the British Athletics Muscle Injury Classification, which incorporated prognostic and diagnostic strength into grading systems.Within this framework, studies were stratified as follows:

High certainty: Evidence derived from well-conducted randomized controlled trials (RCTs) or meta-analyses where further research is very unlikely to change confidence in the findings.

Moderate certainty: Evidence from RCTs with methodological limitations or large prospective cohort studies; further research may have an important impact on confidence.

Low certainty: Evidence from small clinical trials, retrospective cohorts, or studies with significant risk of bias; further research is very likely to change the estimate.

Very low certainty: Evidence based on case series, pilot studies, or expert opinion where the estimate of effect remains highly uncertain.

For clarity, the evidence tables (see Table 1) are colour-coded: green (high), yellow (moderate), orange (low), and red (very low).

Risk of Bias Across Studies

Reported studies demonstrate several recurring sources of bias.[41,42] Rehabilitation protocols are often heterogeneous, making comparison difficult. In regenerative therapies such as platelet-rich plasma (PRP) or stem-cell applications, outcome variability is compounded by differences in preparation methods, dosing, and delivery protocols. Surgical repair trials frequently lack control groups and show variability in follow-up duration, limiting generalisability. Return-to-sport outcomes are particularly vulnerable to measurement bias, as definitions and benchmarks for successful return differ across studies.

Table 1 | Summary of evidence classification for recovery strategies in tendon injuries

Intervention/Strategy	Representative Study Type(s)	Certainty of Evidence (Modified GRADE)	Classification (Colour Code)
Eccentric loading protocols	Multiple Randomized Controlled Trials (RCTs) and meta-analyses	High	Green
Shockwave therapy	RCTs with variable methodological quality	Moderate	Yellow
Platelet-rich plasma (PRP)	Small RCTs, heterogeneous case series	Low–Moderate	Orange / Yellow
Stem-cell therapy (MSCs, PSCs)	Pilot clinical trials, animal studies	Low	Orange
Growth factor therapy (e.g., TGF-β, PDGF)	Preclinical models, limited early-phase trials	Very Low–Low	Red / Orange
Surgical repair (Achilles, rotator cuff, patellar tendon)	Prospective cohorts, some RCTs	Moderate	Yellow
Return-to-sport criteria	Retrospective cohorts, systematic reviews with heterogeneous definitions	Low–Moderate	Orange / Yellow

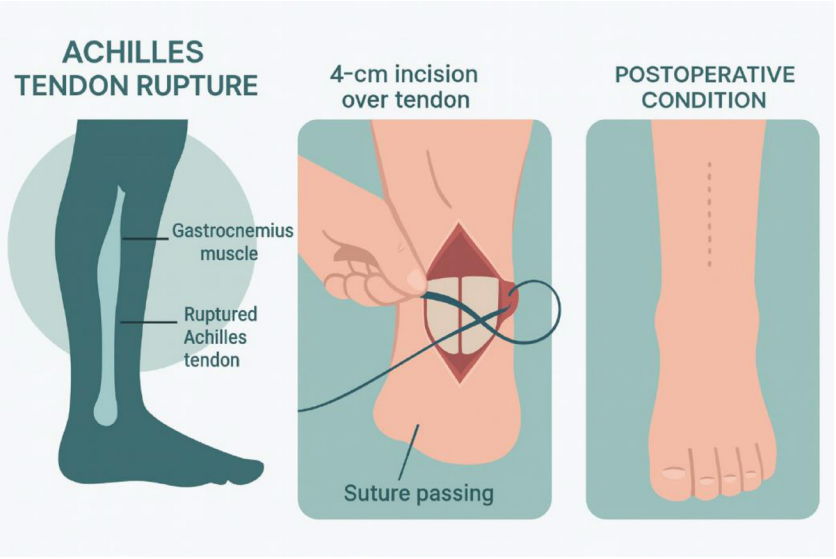

Fig 4 | The illustration demonstrates the stages of Achilles tendon injury and repair. Left: Ruptured Achilles tendon beneath the gastrocnemius muscle. Centre: Surgical repair involving a 4 cm incision and Fiberwire sutures placed through the torn tendon ends. Right: Postoperative condition showing the Achilles tendon reapproximated and skin incision closed

Management of tendon injuries in athletes often begins conservatively, but in cases of complete ruptures, chronic degeneration, or failed non-operative management, surgical intervention becomes necessary. However, despite advances in surgical technique, clinical outcomes remain suboptimal, particularly due to the formation of fibrotic scar tissue and the limited regenerative capacity of tendons. As a result, there is growing interest in biologically based regenerative strategies to enhance tendon healing and improve the durability of surgical repairs.[43]

The Achilles tendon is the body's largest and strongest tendon, essential for powerful movements like sprinting and jumping. It plays a critical role in generating the force needed to push the foot downward during walking or running. Despite its strength and tension, the Achilles tendon is susceptible to injury if overstretched, potentially leading to a tear or complete rupture[44] (Figure 4). These injuries most commonly occur during recreational sports. In cases of rupture, surgical repair may be advised based on an individual's activity level and overall condition. Prompt evaluation by a podiatrist is essential to determine the best treatment approach for Achilles tendinitis or related injuries.

Surgical Repair and Its Limitations

Surgical repair remains the standard of care for complete tendon ruptures and symptomatic partial tears unresponsive to conservative therapy. However, healing frequently results in scar tissue with inferior biomechanical properties, leading to re-rupture or functional limitations. This issue is particularly evident in rotator cuff repairs, where failure rates can be as high as 63% for large and 73% for massive tears despite optimal surgical technique.[43] Intra-articular ligaments, such as the anterior cruciate ligament (ACL), rarely heal spontaneously due to poor vascularisation, necessitating reconstruction with autografts or allografts. While autografts provide good mechanical strength, they are associated with donor site morbidity; allografts, in turn, carry risks of immunogenicity and inferior biomechanical integration.[45]

Growth Factor Therapy

An important area of research has focused on delivering exogenous growth factors to accelerate tendon repair. Molecules such as TGF-β1, PDGF, bFGF, IGF-1, GDF5, and VEGF have all been implicated in the natural healing process and tested experimentally as therapeutic agents.[49] These factors regulate fibroblast proliferation, collagen production, angiogenesis, and matrix remodeling during different phases of tendon healing.[46] Delivery methods include local injection, gene vectors, and biomaterial scaffolds designed for sustained release. Although animal studies have shown some success, especially in the early stages of healing, the long-term efficacy remains variable and inconsistent in human trials.[47]

Platelet-Derived Growth Factor (PDGF)

PDGF plays a central role in the early inflammatory and proliferative phases of tendon healing. It stimulates chemotaxis, fibroblast proliferation, and type I collagen synthesis.[48] PDGF is typically released from activated platelets and can be delivered exogenously through injections or platelet-rich plasma formulations. In preclinical models, PDGF has been shown to enhance tendon cellularity and improve structural organisation.

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However, clinical translation has been limited by short half-life and the need for controlled release systems to avoid overstimulation and fibrotic scarring.

Transforming Growth Factor-Beta (TGF-β)

TGF-β exists in multiple isoforms, with TGF-β1 and TGF-β2 associated with scar formation, while TGF-β3 is linked to regenerative, scarless healing, particularly in fetal tissue.[49] TGF-β1 is a potent driver of extracellular matrix production but may also promote adhesion formation in intrasynovial tendons. Therapeutic manipulation of TGF-β pathways aims to harness its regenerative potential while limiting fibrotic side effects. Attempts to upregulate TGF-β3 or selectively inhibit TGF-β1 have shown some promise in experimental tendon-to-bone healing models but remain technically complex for clinical use.

Basic Fibroblast Growth Factor (bFGF)

bFGF promotes fibroblast proliferation and angiogenesis during the early stages of tendon healing.[50] In animal models, bFGF delivery accelerates cellular infiltration and matrix deposition. However, excessive or prolonged bFGF activity has been associated with disorganised collagen and adhesion formation, particularly in flexor tendon systems. Because of these drawbacks, its therapeutic use is often limited to controlled-release platforms or local delivery in specific injury contexts.

Insulin-like Growth Factor-1 (IGF-1)

IGF-1 has anabolic effects on tendon tissue by enhancing collagen synthesis and promoting tenocyte proliferation.[51] It plays a key role in matrix remodelling and is responsive to mechanical loading, making it particularly relevant to rehabilitation-guided recovery strategies. IGF-1 has demonstrated beneficial effects in improving tendon tensile strength in animal models. However, dosage sensitivity and rapid degradation continue to pose challenges in clinical application.

Growth Differentiation Factor 5 (GDF5)

GDF5 is a member of the bone morphogenetic protein (BMP) family and is involved in tendon and ligament development.[52] It has shown potential in preclinical studies to enhance tendon-to-bone integration and improve mechanical properties at repair sites. GDF5 may also influence stem cell differentiation toward tenogenic lineages. Despite promising early results, clinical data on GDF5 remain sparse, and further trials are needed to establish its therapeutic profile.

Vascular Endothelial Growth Factor (VEGF)

VEGF promotes angiogenesis, which is critical for delivering oxygen and nutrients to healing tendon tissue.[53] Its expression is upregulated during the early inflammatory phase. Controlled VEGF delivery has been shown to improve vascularisation and collagen organisation in tendon injury models. However, uncontrolled VEGF expression may lead to neovascularisation that compromises tendon strength or increases pain, particularly in chronic tendinopathy.

Platelet-Rich Plasma (PRP)

PRP has gained popularity in sports medicine due to its autologous origin and high concentrations of growth factors such as PDGF, VEGF, TGF-β, and EGF.[54–57] It has been investigated in the treatment of rotator cuff tendinopathy, patellar and Achilles tendinopathies, and partial ligament tears. Although in vitro and animal studies show promising outcomes, clinical evidence remains inconsistent. Variability in patient response, PRP formulation (leukocyte-rich vs. leukocyte-poor), dosage, and injection protocols complicate the interpretation of results. Despite the absence of strong evidence, PRP is considered a low-risk adjunctive therapy. However, high costs and lack of standardisation continue to limit its widespread use.[58]

Stem Cell Therapy

Among regenerative approaches, mesenchymal stem cells (MSCs) have demonstrated the greatest promise. Their ability to self-renew, differentiate into tenocytes, and secrete immunomodulatory and trophic factors positions them as strong candidates for tendon repair.[59] MSCs have been sourced from bone marrow (BMSCs), adipose tissue (ASCs), and tendon-derived progenitors (TDSCs). TDSCs, in particular, exhibit superior tenogenic potential compared to other MSCs, but their clinical utility is limited by low harvest yields and the need for in vitro expansion.[60] Therapeutic strategies using MSCs range from direct injection to more advanced tissue engineering constructs, where cells are seeded onto biodegradable scaffolds and combined with growth factors or PRP. These composite systems offer mechanical support and a biologically active matrix to enhance integration and repair. Parameters such as cell type, dose, scaffold composition, and delivery method are critical to efficacy and are being actively optimised in preclinical models.[61]

Emerging research has highlighted the role of perivascular stem cells (PSCs), a subset of MSCs identified by markers such as CD146, Sca-1, and Nestin, that reside around blood vessels and exhibit strong tenogenic potential. PSCs can be harvested in large quantities from adipose tissue, potentially offering a scalable autologous source for therapeutic application. Preliminary studies show that adipose-derived PSCs may match or even surpass TDSCs in tendon healing capacity, though in vivo studies are ongoing.[62]

Vascularisation and Microenvironmental Cues

The vascular supply plays a pivotal role in tendon healing, as illustrated by the stark contrast between the healing capacities of vascularized extra-articular tendons and poorly vascularized intra-articular ligaments. Techniques that enhance vascularity at the repair site, such as bone marrow stimulation, have been shown to improve outcomes in rotator cuff repair.[63] The biological environment, including oxygen tension and inflammatory balance (M1 to M2 macrophage transition), also shapes the quality of tendon remodelling.[64]

Return-to-Sport Criteria

Return to sport (RTS) following tendon injuries represents a critical endpoint in the management of athletic populations. It encompasses not only structural healing but also the restoration of physical performance, psychological readiness, and reintegration into sport-specific demands. Despite advances in surgical and rehabilitative strategies, clear and evidence-based RTS criteria remain inconsistently applied across different tendon injury types.

Overview and Relevance

Tendon rupture, particularly involving the patellar tendon, is a rare but serious injury in athletes, associated with significant loss of knee extensor function and a protracted recovery timeline. RTS outcomes following such injuries are often underreported or poorly defined. A systematic review by Grondin et al. highlighted a lack of standardised criteria for RTS, despite observed return rates ranging from 52% to 100% among 196 patients with 202 patellar tendon ruptures.[65] For instance, M.F. Busa (2025)[66] reported that 25% of professional athletes fail to RTS after Achilles tendon rupture, and those who do RTS show a wide recovery duration (8–11 months), with performance decline up to 50%. These findings were based on a combination of a scoping review of 34 studies, a case series with 23 patients, and a pilot study of 5 football players.

Return Rates and Athletic Level

In studies where pre-injury sport level was documented, professional athletes demonstrated relatively high RTS success. For example, Boublik et al. reported that 22 National Football League players with patellar tendon ruptures returned to sport, and 86% (19/22) resumed at their previous performance level.[67] Marder et al. also observed full RTS in 14 of 15 cases, including both competitive and recreational athletes.[68] Similarly, Kuechle et al. documented 100% RTS in their small cohort, including a high-school athlete and several recreational players.[69] However, not all athletes returned to the same performance level. Maffulli et al. noted that 3 of 8 recreational athletes did not resume sport post-injury, due to persistent pain or psychological discomfort with knee function.[70] These findings highlight that while structural repair may be achieved, subjective and functional barriers can delay or prevent effective return.

Time to Return and Rehabilitation Influence

The time to return to sport (RTS) varies widely depending on injury severity, surgical technique, and rehabilitation protocols. Marder et al. reported an average RTS of 8 months for competitive athletes, compared to 11.5 months for recreational athletes.[68] Other studies cite timelines ranging from 5 to 8 months, with professional athletes typically returning sooner.[71] Early mobilisation is now commonly recommended to minimise stiffness and muscle atrophy; however, it may carry higher complication risks, especially in patients with poor tissue quality or comorbidities.[72] Some studies advocated stationary cycling at 8–12 weeks, progressive loading at 12–20 weeks, and return to jumping or contact sports at a minimum of 6 months post-surgery.[73] However, Serino et al. found that early mobilisation protocols may correlate with increased adverse events, such as tendon re-rupture or the need for revision surgery, especially when protective loading principles are not adequately observed.[72]

Functional and Objective Criteria

Most RTS decisions are made clinically, often relying on subjective functional scores (e.g., Lysholm, Cincinnati, HSS) and clinician judgment. Lysholm scores across studies ranged from 84 to 95, indicating high patient satisfaction and functional recovery in most cases.[73,74] However, these scores often fail to capture dynamic sport-specific performance and psychological readiness. Objective metrics such as range of motion, quadriceps strength, isokinetic testing, and jump tests are increasingly recognised as essential components of RTS criteria but are seldom reported in the literature.[74] Imaging tools like the Insall-Salvati and Caton-Deschamps indices were used to assess patellar position post-repair, but their direct correlation with RTS capacity remains unclear.[75]

A useful framework for understanding return-to-play (RTP) decision-making strategies, particularly to various decision modifiers as reported by Huber et al.[76] Risk Management Decision Theory (RMDT). This general theory of risky decision-making has been adapted to the context of sports, where we modified its core principles to develop a process model specifically tailored to understanding RTP decisions from the athlete's perspective (see Figure 5). The proposed model incorporates key theoretical considerations from RMDT and contextualises them within the unique demands and uncertainties of athletic injury recovery and return.

Limitations and Risk of Bias

This work is a **structured narrative review** supported by a PRISMA-lite study-selection flow; it was **not prospectively registered** and therefore remains vulnerable to selection and publication bias despite transparent methods. We limited inclusion to **English-language** records, which may omit relevant data. Considerable **heterogeneity** exists across tendon sites, competitive levels, diagnostic methods, rehabilitation protocols, biologic formulations, surgical techniques, comparators, and **outcomes** notably non-standardised **return-to-sport** definitions. This heterogeneity, together with small single-centre trials and inconsistent follow-up, **precluded meta-analysis** and increases imprecision and indirectness (particularly for **elite** athletes). Diagnostic accuracy estimates are influenced by operator expertise and variable reference standards; intervention effects may be inflated by **small-study effects** and selective reporting. Where athlete-only evidence was sparse, we included mixed adult populations and clearly labelled them, but **external validity** to high-performance settings is limited. Because formal quantitative risk-of-bias assessment and GRADE

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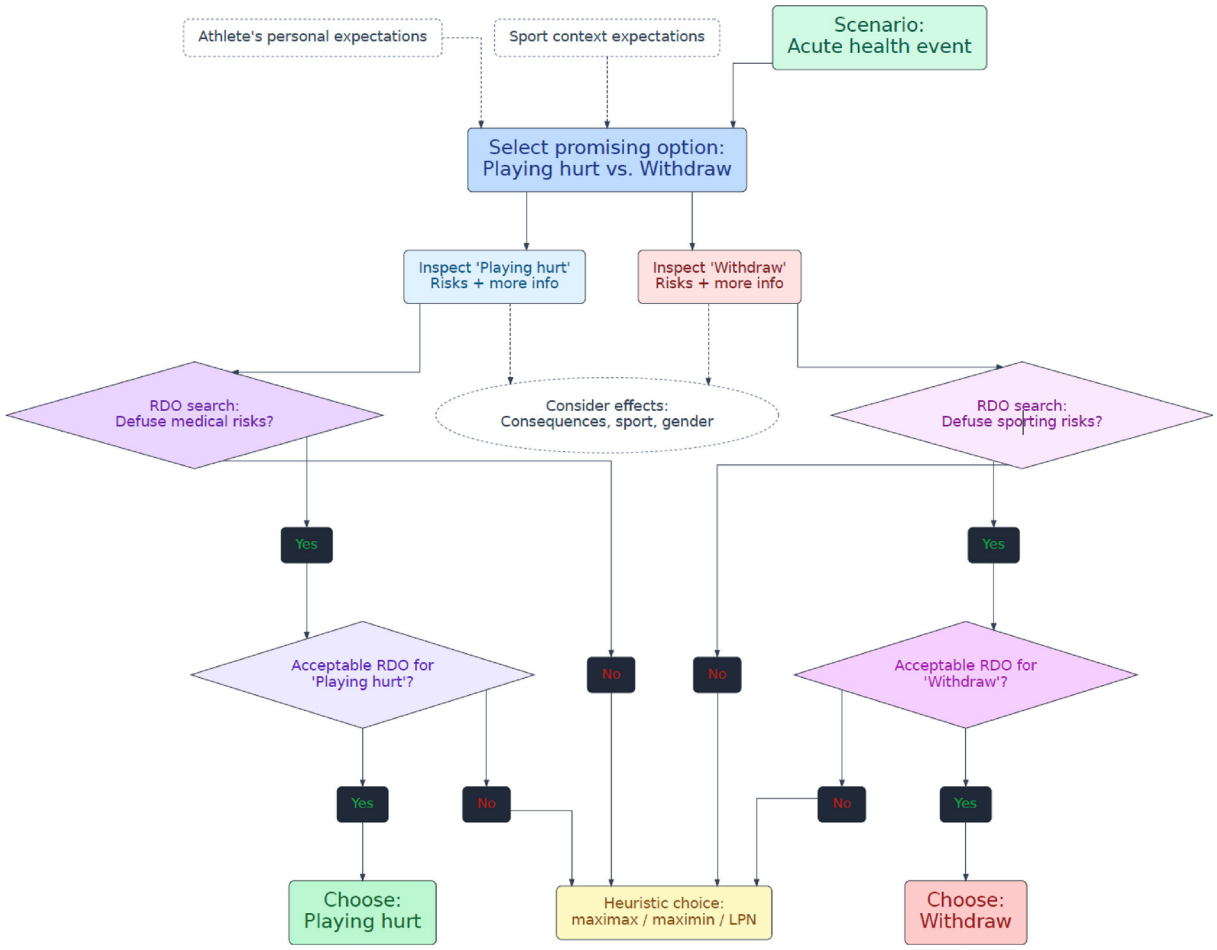

Fig 5 | Process model illustrating return-to-play decision making from the athlete's perspective, adapted from Huber's (2012)[76] Risk Management Decision Theory (RMDT)

rating were not undertaken, we provide **qualitative certainty labels** (higher/moderate/lower/very low) to indicate confidence and we emphasise **ranges** rather than single-point estimates. These limitations should temper interpretation and highlight the need for pre-registered, adequately powered studies using **core outcome sets** and standardised RTS criteria that are sport- and level-specific.

Conclusion

Tendon injuries in athletes represent a complex clinical challenge due to the limited regenerative capacity of tendon tissue, high biomechanical demands in sport, and the multifactorial nature of healing. The pathophysiology of tendon injury involves both intrinsic degeneration and extrinsic overload, with healing proceeding through inflammation, proliferation, and remodelling—yet often resulting in mechanically inferior scar tissue. Variability in healing responses among different tendon types (e.g., rotator cuff vs. Achilles) underscores the need for tendon-specific treatment strategies. Accurate diagnosis and classification remain essential, yet are hampered by the inconsistent sensitivity of clinical tests and non-uniform classification systems. Imaging modalities such as MRA and MRI offer the best diagnostic value for characterising both partial and complete tendon tears. However, standardised use of validated grading systems is still lacking, limiting cross-study comparability and treatment stratification.

Conventional surgical repair continues to be the mainstay for complete ruptures, yet high failure rates, particularly in rotator cuff and patellar tendon repairs, highlight the need for improved biological integration. Regenerative strategies, including growth factor therapy, platelet-rich plasma (PRP), and mesenchymal stem cells (MSCs), are promising adjuncts to enhance intrinsic healing, though clinical translation remains limited by heterogeneity in protocols and outcomes. Tissue engineering approaches, combining cellular therapies with scaffolds, offer an emerging frontier for biologically guided tendon repair.

A major limitation in RTS assessment across tendon injury studies is the lack of standardisation in reporting. Most studies failed to specify the type of sport, pre-injury competitive level, or exact timing and criteria for return. The heterogeneity in rehabilitation protocols and definitions of "return" further complicates comparisons. To address this, future research should adopt validated scales such as the Tegner activity scale or Kelly's scale to quantify sport participation levels pre- and post-injury. Additionally, tracking

psychological readiness, reinjury anxiety, and performance metrics could help refine RTS benchmarks.

Rehabilitation plays a pivotal role in functional recovery, with evidence supporting early but controlled loading to promote organised matrix remodelling. However, protocols vary widely, and there remains no universal consensus on the optimal timing or progression. Return-to-sport (RTS) outcomes after tendon injury are generally favourable, with reported rates between 52% and 100%, but the criteria for clearance often lack objectivity. RTS decisions should incorporate validated functional scores, sport-specific benchmarks, strength testing, and patient-reported outcomes.

As a narrative synthesis, this review is designed to integrate multiple domains of tendon recovery rather than conduct formal comparisons across studies. While it does not generate new quantitative insights, it aims to bridge foundational biology with applied clinical frameworks, highlighting key mechanisms and practices relevant to recovery after tendon injury in athletes. Future work may build on this integration by incorporating systematic data extraction and meta-analytic approaches to refine treatment guidelines and stratify outcomes by injury type and intervention strategy.

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