USOPC TENDINOPATHY EDUCATION MANUAL

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Manual Purpose and Intent

This manual produced by the U.S. Olympic & Paralympic Committee (USOPC) provides evidence-based education on tendinopathy management to both clinicians and high-performance personnel. Whether already well-versed in tendinopathy or simply seeking to establish a baseline of knowledge, this manual should provide both contemporary and clinically relevant information. Additionally, an extensive list of references is included for additional reading where needed.

This manual will cover tendon anatomy and physiology, the pathophysiology of tendinopathy, conceptual load management theories, as well as nutritional and time-loss considerations for the athlete. The manual concludes with tendon-specific rehabilitation sections that aim to provide the reader with practical approaches to managing common types of tendinopathy in high-performing athletes. As this manual is updated, additional resources will be included, via hyperlinks and QR codes where applicable, to provide additional information. These will link to short-form video and media to further enrich the experience of the reader.

The recommendations included within this manual, particularly specific protocols and exercises for certain tendinopathies, are intended to be a guide. The authors acknowledge the information included within this manual may not capture all the individual considerations surrounding a case of tendinopathy. As with any good protocol, the authors appreciate the value of the provider's expertise and clinical reasoning. Given that each athlete is unique with respect to injury history, sport demands, and responsiveness to both modalities and exercise programming, it is recommended that the care team of an athlete take an individualized approach when implementing the recommendations within this manual.

Acknowledgments

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The Healthy Tendon

Anatomy and Physiology of the Tendon

Take Home Points

- Tendons are crucial in linking skeletal muscles to bones, facilitating force transmission, enabling movement, and contributing to joint stabilization.
- The primary cellular unit in tendons is the tenocyte, which is responsible for synthesizing and maintaining connective tissue. Collagen, predominantly Type I, forms the majority of the tendon. Different collagen types with unique structures exist in specialized areas, and the structure of each type determines its functional properties.
- Tendons are hypovascular and hyponeural. The limited blood supply and metabolic rate of tendons decreases their healing rate compared to other soft tissues.

Tendons represent anatomical structures responsible for linking skeletal muscle to bone, thus facilitating the transmission of force to the skeletal system and enabling movement. They also play a crucial role in joint stabilization and the management of potential energy, particularly elastic energy, during kinematic processes.[1] The connection point between a tendon and bone is identified as the osteotendinous junction or enthesis. This junction may take the form of fibrous or fibrocartilaginous structures. In fibrous forms, collagenous tissue directly attaches to the bone, whereas the more common fibrocartilaginous enthesis consists of uncalcified fibrocartilage, calcified fibrocartilage, and bone, and serves as a transitional zone between the tendon and bone.[2–4] The specialized connection between the muscle and tendon is known as the myotendinous junction, where collagen fibers become deeply integrated into the myocyte structure of the skeletal muscle via finger-like projections, incorporating the contractile protein actin[5] and structural proteins such as laminin and dystrophin.[6] This integration permits the tensile force transfer of contractile proteins directly to the collagen fibers of the tendon.

The basic cellular unit of tendons is predominantly the tenocyte. Tenocytes are the primary mediators initiating the intra- and extracellular signaling cascades that regulate collagen remodeling in response to mechanical stress.[7] In addition to tenocytes, ongoing research has revealed the existence of several other cell types,[8] including undifferentiated stem/progenitor cells.[9] These cell types are primarily responsible for synthesizing and maintaining the collagen content of the tendon. The microscopic structure of tendons contains several fibrillar collagen types (collagen that can be aggregated into highly ordered structures - primarily Type I, II, IV - and distinguished from filamentous collagen). Although 28 vertebrae

collagen types have been identified,[10] the most abundant type in tendons is collagen Type I, which can constitute up to 80% of the tendon's dry mass.[11,12] Other collagen types have specialized functions dictated by their unique structures and, thus, are located in varied anatomical structures (Table 1).

Table 1. Primary and specialized functions of the most common human collagen types.

Collagen Type	Structure	Primary Anatomical Location
Type I	Most typically a heterotrimeric helix of Type 1 α1 and Type 1 α2 strands. Homotrimeric structures have been observed in tumors[13] and other pathologies.[10]	Skin, bones, tendons, and ligaments.[14]
Type II	Homotrimeric helix of Type II α1 strands.	Articular (hyaline) cartilage.[15]
Type III	Homotrimeric helix of Type III α1 strands.	Structural component of major organs, vessels, and wound healing. Secreted by fibroblasts thus aiding in scar tissue formation.[16]
Type IV	Heterotrimeric helix of Type IV α1-α6 strands.	Basal lamina.[17]
Type V	Homo- and heterotrimeric helix of Type V α1, α2, and α3 strands.	Commonly present with Type I collagen, dermal/epidermal function, placenta, parachymal organs.[18]

Collagen consists of subunits called tropocollagen, formed by single-stranded peptides known as alpha (α) chains (preprocollagen), which are assembled into a triple-helix polypeptide called procollagen.[19] The addition of hydroxyl groups to specific amino acids on the pre-procollagen permits covalent cross-linkage.[20,21] Critically, this hydroxylation depends on vitamin C as a cofactor; in vitamin C deficiency, the hydroxylation of proline and lysine is limited, thus hindering collagen synthesis, which may result in conditions such as scurvy.[22] Once in the extracellular matrix (ECM), the triple-helix structure is trimmed by peptidases creating tropocollagen, which is subsequently organized into parallel systems via sidechain linkages, forming collagen fibrils up to 500 nm in diameter.[23] Fibrils are then aggregated to form tendon fibers, the smallest visible unit of the tendon (Figure 1). Fibers are aggregated into sub-fascicles encased in a thin connective tissue network (the endotendon). Further bundling (fascicles and tertiary bundles) builds the larger tendon structure, surrounded by the epitendon, facilitating the nerve, vascular, and lymphatic supply.[11] Some tendons, such as the Achilles and quadriceps

tendons and other tendons that are anatomically straight, attach to bone and permit a large degree of motion, can have an additional connective tissue layer (the paratenon), which encases the tendon structure, and assists in extra-tendon tissue interactions. Some tendons, such as at the rotator cuff, lack a paratenon or synovial tendon sheath.[24] The epitenon and paratenon are termed the peritenon and can experience pathological states such as peritenonitis (inflammation of the peritenon).

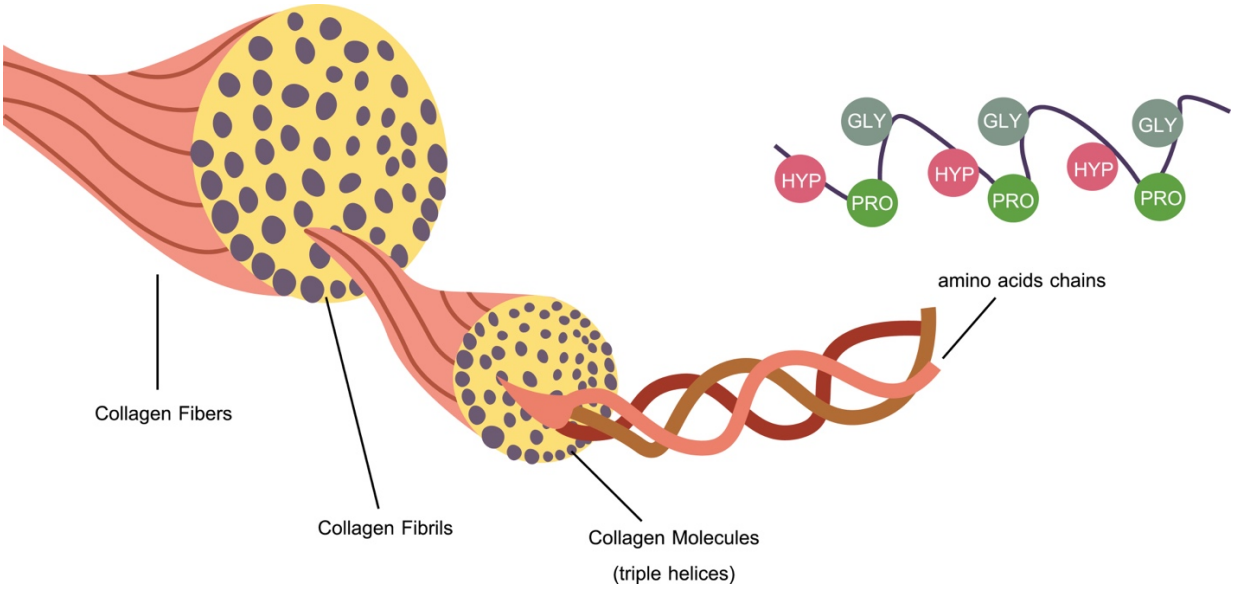

Figure 1. Tendon hierarchy illustrating amino acid chains, the triple helix, collagen molecules, collagen fibrils, and collagen fibers (image obtained from Shutterstock.com).

Tendons tend to be both hypovascular and hyponeural. Blood vessels serving the tendon originate from both the myotendinous and osteotendinous junction, as well as a vascular network around the paratenon, which can penetrate deeper layers of the tendon.[25] The mature tendon tissue consists primarily of extracellular components, have a low metabolic rate and, therefore, do not require a large vascular supply. This, in turn, limits the rate of healing following insult compared to other soft tissues.[26] Neuronally, the limited sensory nerve fiber endings within the tendon can detect changes in tension, pressure, and pain. Specialized sensory receptors (i.e.,Golgi tendon organs) are found at myotendinous junctions, monitor changes in tension and are responsible for inhibitory reflex arcs,[27] although excitatory and inhibitory supraspinal tracts also contribute to this action.[28]

Collagen synthesis following mechanical loading results from a cascade of intracellular signaling pathways, including via integrin-linked kinase and mTOR (mammalian target of rapamycin).[29] Other pathways, such as the ERK1/2 signaling cascades, and a number of growth factors, including insulin-like growth factor-1 (IGF1), transforming growth factor b1 (TGFb1),

and connective tissue growth factor (CTGF), also appear to be critical for tendon growth (see Magnusson and Kjaer[30] for review). In response to injury (e.g. tendinopathy), collagen fibrils tend to appear unorganized,[31] which may be a result of increased water content and vascularization, leading to an increase in cross-sectional area.[30] Innovative research utilizing atmospheric levels of [^{14}C] suggest that tendon collagen is continually developed until approximately age 17, after which collagen synthesis is relatively dormant for the remaining lifespan unless the tendon is injured.[32]

References

1. Labonte D, Holt NC. Elastic energy storage and the efficiency of movement. Curr Biol. 2022;32(12):R661-R666.
2. Apostolakos J, Durant TJ, Dwyer CR, et al. The enthesis: a review of the tendon-to-bone insertion. Muscles Ligaments Tendons J. 2014;4(3):333.
3. Benjamin M, Ralphs J. Entheses--the bony attachments of tendons and ligaments. Ital J Anat Embryol Arch Ital Anat Ed Embriologia. 2001;106(2 Suppl 1):151-157.
4. Benjamin M, Kumai T, Milz S, Boszczyk B, Boszczyk AA, Ralphs JR. The skeletal attachment of tendons—tendon 'entheses.' Comp Biochem Physiol A Mol Integr Physiol. 2002;133(4):931-945.
5. Ciena AP, Luques IU, Dias FJ, de Almeida SRY, Iyomasa MM, Watanabe I sei. Ultrastructure of the myotendinous junction of the medial pterygoid muscle of adult and aged Wistar rats. Micron. 2010;41(8):1011-1014.
6. Charvet B, Ruggiero F, Le Guellec D. The development of the myotendinous junction. A review. Muscles Ligaments Tendons J. 2012;2(2):53.
7. Khan KM, Scott A. Mechanotherapy: how physical therapists' prescription of exercise promotes tissue repair. Br J Sports Med. 2009;43(4):247-252.
8. Wu F, Nerlich M, Docheva D. Tendon injuries: Basic science and new repair proposals. EFORT Open Rev. 2017;2(7):332-342.
9. Bi Y, Ehirchiou D, Kilts TM, et al. Identification of tendon stem/progenitor cells and the role of the extracellular matrix in their niche. Nat Med. 2007;13(10):1219-1227.
10. Holmes DF, Lu Y, Starborg T, Kadler KE. Collagen fibril assembly and function. Curr Top Dev Biol. 2018;130:107-142.
11. Sharma P, Maffulli N. Biology of tendon injury: healing, modeling and remodeling. J Musculoskelet Neuronal Interact. 2006;6(2):181.
12. O'brien M. Structure and metabolism of tendons. Scand J Med Sci Sports. 1997;7(2):55-61.
13. Pucci-Minafra I, Andriolo M, Basirico L, et al. Absence of regular alpha2 (I) collagen chains in colon carcinoma biopsy fragments. Carcinogenesis. 1998;19(4):575-584.
14. Bou-Gharios G, de Crombrugghe B. Type I Collagen Structure, Synthesis, and Regulation. Princ Bone Biol. Published online 2008:285.
15. Eyre D. Articular cartilage and changes in arthritis: collagen of articular cartilage. Arthritis Res Ther. 2001;4:1-6.
16. Kuivaniemi H, Tromp G. Type III collagen (COL3A1): Gene and protein structure, tissue distribution, and associated diseases. Gene. 2019;707:151-171.
17. Steffensen LB, Rasmussen LM. A role for collagen type IV in cardiovascular disease? Am J Physiol-Heart Circ Physiol. 2018;315(3):H610-H625.

18. Mak KM, Png CYM, Lee DJ. Type V collagen in health, disease, and fibrosis. Anat Rec. 2016;299(5):613-629.
19. Silver FH, Freeman JW, Seehra GP. Collagen self-assembly and the development of tendon mechanical properties. J Biomech. 2003;36(10):1529-1553.
20. Kadler KE, Holmes DF, Trotter JA, Chapman JA. Collagen fibril formation. Biochem J. 1996;316(1):1-11.
21. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. Nat Rev Rheumatol. 2010;6(5):262-268.
22. Peterkofsky B. Ascorbate requirement for hydroxylation and secretion of procollagen: relationship to inhibition of collagen synthesis in scurvy. Am J Clin Nutr. 1991;54(6):S1135-S1140.
23. Van Der Rijt JA, Van Der Werf KO, Bennink ML, Dijkstra PJ, Feijen J. Micromechanical testing of individual collagen fibrils. Macromol Biosci. 2006;6(9):697-702.
24. Gott M, Ast M, Lane LB, et al. Tendon phenotype should dictate tissue engineering modality in tendon repair: a review. Discov Med. 2011;12(62):75-84.
25. Ahmed I, Lagopoulos M, McConnell P, Soames R, Sefton G. Blood supply of the Achilles tendon. J Orthop Res. 1998;16(5):591-596.
26. Williams J. Achilles tendon lesions in sport. Sports Med. 1986;3:114-135.
27. Eccles J, Eccles RM, Lundberg A. Synaptic actions on motoneurones caused by impulses in Golgi tendon organ afferents. J Physiol. 1957;138(2):227.
28. Moore JC. The Golgi tendon organ: a review and update. Am J Occup Ther Off Publ Am Occup Ther Assoc. 1984;38(4):227-236. doi:10.5014/ajot.38.4.227
29. Mousavizadeh R, Hojabrpour P, Eltit F, et al. β1 integrin, ILK and mTOR regulate collagen synthesis in mechanically loaded tendon cells. Sci Rep. 2020;10(1):12644.
30. Magnusson SP, Kjaer M. The impact of loading, unloading, ageing and injury on the human tendon. J Physiol. 2019;597(5):1283-1298.
31. Pingel J, Wienecke J, Kongsgaard M, et al. Increased mast cell numbers in a calcaneal tendon overuse model. Scand J Med Sci Sports. 2013;23(6):e353-e360.
32. Heinemeier KM, Schjerling P, Heinemeier J, Magnusson SP, Kjaer M. Lack of tissue renewal in human adult Achilles tendon is revealed by nuclear bomb 14C. FASEB J. 2013;27(5):2074.

Basic Science and Conceptual Load Management Theories of Tendinopathy

Take Home Points

- Originally proposed in 1996, Dr. Scott Dye's *Envelope of Function* is a theory that represents the functional capacity of a specific joint in accepting, transferring, or dissipating load.[1] The Physical Stress Theory, published in 2002, proposes a similar model expanded to represent all biological tissue.[2]
- A tissue progressively loaded over time will demonstrate a higher envelope of function, suggesting the tissue is better able to withstand and tolerate load.[1] Conversely, a tissue without exposure to regular load will atrophy and become less tolerant of physical stresses.[1,2]
- A 2-to-3-fold increase in tendon collagen formation peaks around 24 hours post exercise and remains elevated for 70 to 80 hours.[3,4] In addition, the degradation of collagen proteins post exercise also increases.[4] After 72 hours, the net effect of these competing factors ends in a net positive of synthesis. Without sufficient rest (24 hours) post exercise, there may be a net loss of collagen that leaves the tendon vulnerable to injury.[5]
- Although different theories exist, the onset of symptoms associated with tendinopathy (i.e., the "metabolic tipping point") is thought to indicate late-stage tendinopathic disease.[6,7]

In clinical practice, monitoring training load provides performance and medical staff an opportunity to potentially estimate individualized injury risk. Over the last several decades of sport, a considerable amount of focus has been placed on better understanding the relationship between load and injury.[9,10] As a result, coaching staff are better informed and able to finely tune practice volumes and intensity.

The Physical Stress Theory, first published in 2002, states that changes in the relative level of physical stress cause a predictable adaptive response in all biological tissue.[2] Physical stress is the force, or load, acting on a given area of tissue.[2,8] This theory proposes that tissues accommodate to physical stresses by altering their structure and composition to best meet the mechanical demands of routine loading.[2] Deviations from an individual's routine loading, whether it's the addition or removal of load, provide a stimulus to tissue adaptation.[2]

The "adaptive capacity" of a tissue, when exceeded, results in tissue injury or death.[2] The term adaptive capacity has been described across multiple different publications with synonymous naming such as "tissue capacity"[11] or the "Envelope of Function."[1] Regardless of the

preferred name, a tendon is at full capacity when it can be used to perform functional movements at the required volume and frequency (typically in sport) without exacerbating symptoms or causing injury.[11] When a tissue's capacity is exceeded, either in volume or training intensity, pain or injury may arise. Thankfully, tissue capacity can be improved with progressive and appropriate loading (i.e., rehabilitation and strength and conditioning).

This concept was described in Dye's Envelope of Function.[1] This theory attempts to represent the tissue capacity of a specific joint or tissue in accepting, transferring, or dissipating loads. In this model, one can consider the frequency and magnitude of load applied to a tissue. Naturally, various activities will carry different load profiles, exposing a tendon to a variety of stresses.[1] Imagine a theoretical example of the envelope of function for the Achilles tendon of a healthy, young athlete. One can consider activities such as walking 10 km to have a high frequency of loading but a relatively low magnitude of loading on the Achilles tendon, whereas 2 hours of soccer have a relatively larger magnitude load – however, both activities exist below the envelope of function.

One can now consider the impact on the same tendon following rupture and subsequent surgical repair – namely, that the envelope of function becomes greatly diminished following injury. Consequently, activities that were previously well tolerated now would exceed the new envelope of function and may be more challenging and symptomatic. Of course, in this example, the Achilles tendon can be reconstructed, and following extensive rehabilitation, the envelope of function can be improved again. This model serves as an excellent conceptualization of the basic rehabilitative management of tendinopathy.

An injured tendon with a low envelope of function must be mechanically loaded to adapt. Rehabilitative exercises, or strength programming, should occur at loads marginally above the envelope of function or in the supraphysiological overload zone. For intended adaptations (e.g., improved strength, improved tolerance) to occur, tissues must be stressed in a specific manner.[12] Should the load applied be less than the envelope of function and fall into the zone of homeostasis, rehabilitative benefits or performance improvements generally will not occur. Whilst not always quantifiable, clinicians and practitioners should be constantly evaluating and updating their conceptualization of an athlete's envelope of function, attempting to load the athlete or specific tissues within the zone of supraphysiological overload. In turn, the desired compensatory physiological adaptation is reached without overextending the load into the zone of structural overload where athletes are less likely to show positive adaptation and may experience exacerbations in symptoms.

As these theories imply, tendons are metabolically active in their response to loading.[13,14] From a biological perspective, applying mechanical load to tendon results in an upregulation of

collagen expression and increased synthesis of collagen protein in both animals and humans.[3,5,15] This increase in collagen expression is likely regulated by the strain applied on the tenocyte, which is the resident fibroblast of a tendon.[5] Fibroblasts are diverse mesenchymal cells that assist in tissue homeostasis and disease through the production of complex extracellular matrix and creation of signaling niches through both biophysical and biochemical cues.[16] After exercise, a 2 to 3 fold increase in collagen formation peaks around 24 hours post exercise and remains elevated for 70 to 80 hours.[3,4] There is also an increase in collagen protein degradation post exercise;[4] however, the end result is a net positive synthesis after 72 hours.[5]

From a molecular perspective, exercise results in an increase of mRNA levels and in tissue concentrations of growth factors such as insulin-like growth factor (IGF-1), transforming growth factor β (TGF-β), connective tissue growth factor (CTGF) and interleukin (IL)-6.[5] IGF-1 is known as a key regulator of collagen synthesis,[17,18] while TGF-β and CTGF stimulate fibroblasts.[19] Habitual loading, of which the majority of athletes participating in sport are exposed, will result in a higher rate of collagen synthesis in the basal state due to the constant effect of the previous 24 to 48 hours.[20] Without sufficient rest (24 hours) post exercise, net loss of collagen may occur that leaves the tendon vulnerable to injury.[5]

Interestingly, recent publications have described the repair capacity of a tendon to be deeply linked to its complex physiological interplay between an "intrinsic compartment" and "extrinsic compartment."[21-23] The intrinsic tendon compartment comprises the fibrous collagen core and the extrinsic tendon compartment consists of the surrounding synovial-like tissues.[21-23] In chronic tendinopathy, Snedeker & Foolen propose that a progressive accumulation of intrinsic tissue damage occurs until the tendon core reaches a "metabolic tipping point."[6] They speculate that the tipping point is reached when the metabolic demands of the tendon core exceed the available nutrient supply of the normally avascular core.[6,24] From here, the extrinsic tendon compartment is recruited by the tendon core to assist in organ / tissue remodeling, possibly driving additional recruitment of vasculature and nerve supply to the tendon core,[6,25] both of which are observed commonly in chronic tendon disease. Snedeker & Foolen further propose that until the metabolic tipping point of a tendon is reached, the patient may be entirely pain free. The tipping point marks the moment that intrinsic repair mechanisms are overwhelmed and that the neovascularization and ingrowth of innervation contribute to typical symptoms seen in tendinopathy.[6]

Carbon-14 ([^{14}C]) bomb pulse testing, a specialized type of testing used to date organic materials by measuring the elevated levels of [^{14}C] in the atmosphere caused by nuclear weapon testing between 1955 and 1963, has determined the bulk of collagen matrix in a healthy human Achillies tendon core is essentially a permanent structure that is laid during height growth with

limited turnover in adults.[26] A more recent publication by Heinemeier et al, using the same carbon dating method, suggests that substantial collagen renewal occurs in the tendinopathic tendon.[7] Similar to Snedeker & Foolen's work, Heinemeier et al. suggested that symptoms of tendinopathy may represent a very late stage of a tendinopathic disease process, or possibly that a high collagen exchange could be a risk factor for tendon disorders rather than the sequalae of tendinopathy itself.[7]

The clinician should seek to understand tendinopathy management not only from a conceptual load management model,[1,2] but also from a basic science perspective as well.[5-7] An appreciation from both angles ultimately helps to guide initial evaluation and rehabilitative programming that compliments the daily, weekly, and monthly demands of the athlete.

References

1. Dye SF. The Knee as a Biologic Transmission With an Envelope of Function. Clinical Orthopaedics and Related Research. 1996;(323):10-18.
2. Mueller MJ, Maluf KS. Tissue adaptation to physical stress: A proposed “Physical Stress Theory” to guide physical therapy practice, education, and research. Phys Ther. 2002;82(4):383-403.
3. Miller BF, Olesen JL, Hansen M, et al. Coordinated collagen and muscle protein synthesis in human patella tendon and quadriceps muscle after exercise. J Physiol. 2005;567(Pt 3):1021-1033.
4. Langberg H, Skovgaard D, Karamouzis M, Bülow J, Kjaer M. Metabolism and inflammatory mediators in the peritendinous space measured by microdialysis during intermittent isometric exercise in humans. J Physiol. 1999;515 (Pt 3)(Pt 3):919-927.
5. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. Nat Rev Rheumatol. 2010;6(5):262-268.
6. Snedeker JG, Foolen J. Tendon injury and repair - A perspective on the basic mechanisms of tendon disease and future clinical therapy. Acta Biomater. 2017;63:18-36.
7. Heinemeier KM, Schjerling P, Øhlenschlæger TF, Eismark C, Olsen J, Kjær M. Carbon-14 bomb pulse dating shows that tendinopathy is preceded by years of abnormally high collagen turnover. FASEB J. 2018;32(9):4763-4775.
8. Mueller MJ. Therapeutic footwear helps protect the diabetic foot. J Am Podiatr Med Assoc. 1997;87(8):360-364.
9. Gabbett TJ. How Much? How Fast? How Soon? Three Simple Concepts for Progressing Training Loads to Minimize Injury Risk and Enhance Performance. J Orthop Sports Phys Ther. 2020;50(10):570-573.
10. Eckard TG, Padua DA, Hearn DW, Pexa BS, Frank BS. The relationship between training load and injury in athletes: A systematic review. Sports Med. 2018;48(8):1929-1961.
11. Cook JL, Docking SI. “Rehabilitation will increase the ‘capacity’ of your …insert musculoskeletal tissue here….” Defining ‘tissue capacity’: a core concept for clinicians. Br J Sports Med. 2015;49(23):1484-1485.
12. Baechle, TR, Earle, RW, and Wathen, D. Essentials of Strength Training and Conditioning (3rd Edition). Champaign, IL: Human. Human Kinetics; 2008.
13. Bojsen-Møller J, Kalliokoski KK, Seppänen M, Kjaer M, Magnusson SP. Low-intensity tensile loading increases intratendinous glucose uptake in the Achilles tendon. J Appl Physiol. 2006;101(1):196-201.
14. Langberg H, Rosendal L, Kjær M. Training-induced changes in peritendinous type I collagen turnover determined by microdialysis in humans. J Physiol. 2001;534(1):297-302.

15. Heinemeier KM, Olesen JL, Haddad F, et al. Expression of collagen and related growth factors in rat tendon and skeletal muscle in response to specific contraction types. J Physiol. 2007;582(Pt 3):1303-1316.
16. Plikus MV, Wang X, Sinha S, et al. Fibroblasts: Origins, definitions, and functions in health and disease. Cell. 2021;184(15):3852-3872.
17. Kjaer M, Langberg H, Heinemeier K, et al. From mechanical loading to collagen synthesis, structural changes and function in human tendon. Scand J Med Sci Sports. 2009;19(4):500-510.
18. Abrahamsson SO. Similar effects of recombinant human insulin-like growth factor-I and II on cellular activities in flexor tendons of young rabbits: experimental studies in vitro. J Orthop Res. 1997;15(2):256-262.
19. Schild C, Trueb B. Mechanical stress is required for high-level expression of connective tissue growth factor. Exp Cell Res. 2002;274(1):83-91.
20. Couppé C, Kongsgaard M, Aagaard P, et al. Habitual loading results in tendon hypertrophy and increased stiffness of the human patellar tendon. J Appl Physiol. 2008;105(3):805-810.
21. Liu CF, Aschbacher-Smith L, Barthelery NJ, Dyment N, Butler D, Wylie C. What we should know before using tissue engineering techniques to repair injured tendons: a developmental biology perspective. Tissue Eng Part B Rev. 2011;17(3):165-176.
22. Ackermann PW, Salo P, Hart DA. Tendon innervation. Adv Exp Med Biol. 2016;920:35-51.
23. Ackermann PW. Neuronal pathways in tendon healing and tendinopathy - update. Front Biosci. 2014;19(8):1251.
24. Ackermann PW, Hart DA. General overview and summary of concepts regarding tendon disease topics addressed related to metabolic disorders. Adv Exp Med Biol. 2016;920:293-298.
25. Rio E, Moseley L, Purdam C, et al. The pain of tendinopathy: physiological or pathophysiological? Sports Med. 2014;44(1):9-23.
26. Heinemeier KM, Schjerling P, Heinemeier J, Magnusson SP, Kjaer M. Lack of tissue renewal in human adult Achilles tendon is revealed by nuclear bomb (14)C. FASEB J. 2013;27(5):2074-2079.

Tendon Injury Prevention

Take Home Points

- The majority of general injury prevention strategies appear to also have benefits for tendons (dynamic warm-ups, tailored exercise programming, load management strategies).
- Some studies exist that have specifically evaluated injury prevention programs for site-specific tendinopathies (proximal hamstring, patellar, and Achilles tendons). While results are mixed, there appears to be a protective benefit with tailored exercise programming.
- Passive therapeutic modalities tend to have little scientific evidence of preventing tendon injury (low level laser treatment, iontophoresis, phonophoresis, therapeutic ultrasound, deep friction massage, or general massage).

Muscle power output and efficiency during contractions are dependent on the interaction of the muscle with its mechanical environment.[1] Muscle tissue interacts with bone through the muscle-tendon interface, which is an anatomically specialized region involved in the efficient transmission of force,[2] in turn, producing motion. Tendons can store 400 to 1,800 times more elastic strain energy per unit mass than muscle,[3] making their function and health paramount in sport and activities of daily living. This energy storage and release significantly decreases the energy needed to produce movement.[4]

Dynamic Warm-Ups

Physical performance tends to decline with lowered body and muscle temperature.[5,6] In relation to the tendon specifically, lower temperatures generally increase the stiffness of tendons,[7] likely affecting compliance of the tendon. In both training and competition, a warm-up routine has been suggested to be critical in increasing preparedness for subsequent effort and maximizing performance.[8] Exercise selection for a warm-up should be sport-specific, promote flexibility, exert enough effort on the musculoskeletal system to raise body temperature, and prime the connective tissue and nervous system.[9] Although there are a range of specific and theoretically sound coaching philosophies, a shorter warm-up period of 10 to 15 minutes appears to result in better explosive performance than traditional warm-up routines currently used in sport.[6] In passive transitions longer than 15 minutes (e.g., athletes waiting upon entry into the game, returning to the field of play after a half, period, etc.) a 2-minute short-term explosive task allows the recovery of performance levels achieved during an initial warm-up.[6]

Tailored Exercise Programming

In addition to the implementation of dynamic warm-ups in sport, tailored exercise programming is a popular intervention to ideally decrease the incidence of tendinopathy. Although some data suggests that prophylactic eccentric training and stretching increases the risk of tendon injury,[10] other cohort studies have shown tailored exercise programming to reduce injury risk of patellar tendinopathy by 51%.[11] A sport-specific training program in soccer players found a dose-effect relationship between the duration of "protective" balance training and incident rate of hamstring, patellar and Achilles tendinopathy.[12] The authors described these drills as "protective" balance training drills which included multi-studded soccer shoes on a variety of surfaces to improve athlete proprioception.[12] A landmark 2019 publication study across 35 semiprofessional Norwegian football teams explored the effects of a simple adductor strengthening program, 3 times a week, during the competitive season and saw the prevalence and risk of groin problems fall by 41%.[13]

These exercise training programs should be specific to the demands of the sport and the incidence and prevalence of targeted tendinopathies within the population of interest. For more exercise programming specifics, please refer to the tendinopathy specific sections at the end of this manual including patellar tendinopathy, Achilles tendinopathy, adductor tendinopathy, proximal hamstring tendinopathy, and rotator cuff tendinopathy.

Passive Therapeutic Modalities

In general, there is presently little evidence available to support the use of most passive modalities in the treatment of tendinopathy including low level laser treatment, iontophoresis, phonophoresis, therapeutic ultrasound, deep friction massage, or general massage.[14] Most passive modalities have not been studied for their effectiveness of injury prevention. Traditionally, massage has been thought to benefit athletes by enhancing performance and improving recovery.[15] Unfortunately, there is little, or no, scientific data supporting massage as an effective injury or tendinopathy prevention strategy.[16,17] Instrument assisted soft tissue massage has been shown in some studies to stimulate tendons, but to what extent structural and functional changes in tendons influence prevention strategies are unclear.[18]

Extracorporeal shockwave therapy (ESWT) has grown in popularity since its musculoskeletal inception in the treatment of plantar fasciitis in 1996.[19] It has since been studied in Achilles, patellar, hamstring, greater trochanteric pain syndrome, lateral epicondylitis and tendinopathy of the shoulder with generally mixed results.[20] Growing literature suggests that ESWT may be effective in treating a subset of chronic tendon and plantar fascia[21] diseases in a subset of patients.[22] A recent 2024 systematic review found ESWT may offer an efficacious

treatment alone or as an adjunct to exercise therapy in athlete and physically active individuals.[23] Additional high-level research is needed.

Load Management

The monitoring, quantification and reporting of player workloads has gained significant popularity over the last two decades.[24] While acute to chronic workload ratios (a measurement made in an effort to quantify injury risk) have been challenged extensively,[25–27] associated general themes are typically accepted in load monitoring and prospective periodization by performance, rehabilitative and coaching staff. Three fundamental concepts are critical in developing rehabilitative or performance programs: the "floor," the "ceiling" and time.[28] The floor represents the athlete's (or tendon of interest's) starting capacity and the ceiling represents the athlete's capacity needed to perform his or her sport. The time represents the duration (days, weeks, months) that an athlete and provider have in order progressively expose, or overload, the athlete prior to competition.[28]

Although some of the literature referenced in this section is not "tendon specific," these principles may generally help guide the clinician to discourage the likelihood of injury (including the development of tendinopathies). In summary,[28] the athlete (or tendon of interest) can globally benefit from the following basic principles:

- Maintain an adequate training load during the offseason[24] to maintain an acceptable "floor."
- Identify the ceiling and ensure training load is proportionate to competition demands.[29]
- Assess individual difference in training tolerance among athletes.[28]
- Identify and prepare for the most demanding passages of play[30,31] or high-risk periods of the season.[32]
- Training programs should reflect the physical demands of sport, physical capacities required to perform the sport and factors that limit performance on an individual basis.[28]

References

1. Lichtwark GA, Barclay CJ. The influence of tendon compliance on muscle power output and efficiency during cyclic contractions. J Exp Biol. 2010;213(5):707-714.
2. Narayanan N, Calve S. Extracellular matrix at the muscle - tendon interface: functional roles, techniques to explore and implications for regenerative medicine. Connect Tissue Res. 2021;62(1):53-71.
3. Alexander R, Bennet-Clark HC. Storage of elastic strain energy in muscle and other tissues. Nature. 1977;265:114-117.
4. Benjamin M, Ralphs JR. Tendons and ligaments: An Overview. Histol Histopathol. 1997;12:1135-1144.
5. Racinais S, Oksa J. Temperature and neuromuscular function. Scand J Med Sci Sports. 2010;20 Suppl 3:1-18.
6. Silva LM, Neiva HP, Marques MC, Izquierdo M, Marinho DA. Effects of Warm-Up, Post-Warm-Up, and Re-Warm-Up Strategies on Explosive Efforts in Team Sports: A Systematic Review. Sports Med. 2018;48(10):2285-2299.
7. Hunter J, Kerr EH, Whillans MG. The relation between joint stiffness upon exposure to cold and the characteristics of synovial fluid. Can J Med Sci. 1952;30(5):367-377.
8. McCrary JM, Ackermann BJ, Halaki M. A systematic review of the effects of upper body warm-up on performance and injury. Br J Sports Med. 2015;49(14):935-942.
9. Smith CA. The warm-up procedure: To stretch or not to stretch. A brief review. JOSPT. 1994;19(1):12-17.
10. Fredberg U, Bolvig L, Andersen NT. Prophylactic training in asymptomatic soccer players with ultrasonographic abnormalities in Achilles and patellar tendons: the Danish Super League Study. Am J Sports Med. 2008;36(3):451-460.
11. Bittencourt NFN, Oliveira RR de, Vaz R de PM, Silva RS, Mendonça LDM. Preventive effect of tailored exercises on patellar tendinopathy in elite youth athletes: A cohort study. Phys Ther Sport. 2022;53:60-66.
12. Kraemer R, Knobloch K. A soccer-specific balance training program for hamstring muscle and patellar and achilles tendon injuries: an intervention study in premier league female soccer. Am J Sports Med. 2009;37(7):1384-1393.
13. Harøy J, Clarsen B, Wiger EG, et al. The adductor strengthening programme prevents groin problems among male football players: a cluster-randomised controlled trial. Br J Sports Med. 2019;53(3):150-157.
14. Andres BM, Murrell GAC. Treatment of tendinopathy: what works, what does not, and what is on the horizon. Clin Orthop Relat Res. 2008;466(7):1539-1554.

15. Thomas K. Evidence for instrument-assisted massage. Co-Kinetic Journal. 2023;95:27-32.
16. Gasibat Q, Suwehli W. Determining the benefits of massage mechanisms: A review of literature. Rehabilitation Science. 2017;2(3):58-67.
17. Davis HL, Alabed S, Chico TJA. Effect of sports massage on performance and recovery: a systematic review and meta-analysis. BMJ Open Sport Exerc Med. 2020;6(1):e000614.
18. Kim J, Sung DJ, Lee J. Therapeutic effectiveness of instrument-assisted soft tissue mobilization for soft tissue injury: mechanisms and practical application. J Exerc Rehabil. 2017;13(1):12-22.
19. Rompe JD, Hopf C, Nafe B, Burger R. Low-energy extracorporeal shock wave therapy for painful heel: a prospective controlled single-blind study. Arch Orthop Trauma Surg. 1996;115(2):75-79.
20. Reilly JM, Bluman E, Tenforde AS. Effect of shockwave treatment for management of upper and lower extremity musculoskeletal conditions: A narrative review. PM R. 2018;10(12):1385-1403.
21. Lou J, Wang S, Liu S, Xing G. Effectiveness of extracorporeal shock wave therapy without local anesthesia in patients with recalcitrant plantar fasciitis: A meta-analysis of randomized controlled trials. Am J Phys Med Rehabil. 2017;96(8):529-534.
22. Wang CJ. Extracorporeal shockwave therapy in musculoskeletal disorders. J Orthop Surg Res. 2012;7(1):11.
23. Rhim HC, Shin J, Kang J, et al. Use of extracorporeal shockwave therapies for athletes and physically active individuals: a systematic review. Br J Sports Med. 2024;58(3):154-163.
24. Blanch P, Gabbett TJ. Has the athlete trained enough to return to play safely? The acute:chronic workload ratio permits clinicians to quantify a player's risk of subsequent injury. Br J Sports Med. 2016;50(8):471-475.
25. Impellizzeri FM, Woodcock S, Coutts AJ, Fanchini M, McCall A, Vigotsky AD. What Role Do Chronic Workloads Play in the Acute to Chronic Workload Ratio? Time to Dismiss ACWR and Its Underlying Theory. Sports Med. 2021;51(3):581-592.
26. Impellizzeri FM, Woodcock S, Coutts AJ, Fanchini M, Vigotsky A. Acute to random workload ratio is 'as' associated with injury as acute to actual chronic workload ratio: time to dismiss ACWR and its components. Published online March 5, 2020. doi:10.31236/osf.io/e8kt4
27. Impellizzeri FM, Tenan MS, Kempton T, Novak A, Coutts AJ. Acute:Chronic Workload Ratio: Conceptual Issues and Fundamental Pitfalls. Int J Sports Physiol Perform. Published online June 5, 2020:1-7.
28. Gabbett TJ. How Much? How Fast? How Soon? Three Simple Concepts for Progressing Training Loads to Minimize Injury Risk and Enhance Performance. J Orthop Sports Phys Ther. 2020;50(10):570-573.
29. Drew MK, Cook J, Finch CF. Sports-related workload and injury risk: simply knowing the risks will not prevent injuries: Narrative review. Br J Sports Med. 2016;50(21):1306-1308.

30. Spencer M, Lawrence S, Rechichi C, Bishop D, Dawson B, Goodman C. Time-motion analysis of elite field hockey, with special reference to repeated-sprint activity. J Sports Sci. 2004;22(9):843-850.
31. Gabbett TJ, Jenkins DG, Abernethy B. Physical demands of professional rugby league training and competition using microtechnology. J Sci Med Sport. 2012;15(1):80-86.
32. Anderson T, Wasserman EB, Shultz SJ. Anterior Cruciate Ligament Injury Risk by Season Period and Competition Segment: An Analysis of National Collegiate Athletic Association Injury Surveillance Data. J Athl Train. 2019;54(7):787-795.

Tendon-Specific Loading and Movement Pattern Considerations

Take Home Points

- Tenocytes, the primary cell in tendon, are mechanosensitive, meaning they respond to mechanical stress.
- Loading patterns will vary based upon the anatomy and function of the tendon.
- All muscle contraction types are beneficial when managing tendinopathy with types to be programmed based upon the region and healing status of the involved tendon.
- Hip dominant movement may be utilized when knee-dominant loads exacerbate patellar tendinopathy symptoms.
- The clinician should differentiate between Achilles insertional tendinopathy versus midsubstance tendinopathy. Achilles insertional tendinopathy is more likely to be aggravated when the tendon is loaded in dorsiflexion over the edge of an elevated surface.
- Hip flexion angles need to be considered when programming targeted exercises for proximal hamstring tendinopathy.

Mechanotransduction, the mechanism by which mechanical stress influences genetic and cellular behavior,[1] is a key mechanism at play when rehabilitating the injured tendon. The primary cell type in tendons, tenocytes, are mechanosensitive, with specific responses to underloading, physiologic loading, and overloading.[1–4] Given the direct therapeutic effects of loading, exercise therapies are one of the most consistently effective treatments across all tendinopathies.[5–10] While eccentrics have traditionally been celebrated as the most successful contraction type in the management of tendinopathy,[10] other publications have supported that exercise and contraction type may be individualized based on patient response, tolerance and staging.[11–13] Contraction considerations in programming will likely depend more on staging and irritability. For example, submaximal isometrics are typically tolerated acutely, while plyometric programming is typically tolerated best in “late-stage” rehabilitation. Naturally, clinicians must select exercises that target and apply load to the tendon of interest. Some common loading strategies and movement considerations are outlined below.

Hip Dominant versus Knee Dominant Loading Patterns in Patellar Tendinopathy

The squat (Figure 1), a commonly used exercise in both performance training and rehabilitation alike,[14], can be individualized depending on the tendon of interest. Specific to the

knee and patellar tendon loading, the squat can either be coached to keep the tibial shank as vertical as possible, or to let the knees pass the toes as much as preferred by the athlete. Significantly less torque is produced at the knees when the shank is coached to stay vertical, while significantly higher torque is produced at the knees when the shank is allowed to translate anteriorly over the toes.[15]

Figure 1. A) Allowing the knees to translate forward produces more torque at the knees.[15] An example of this would be a front squat. B.) Keeping the shank more vertical produces more torque at the hips.[15] An example of this would be a banded "Basas Spanish" Squat[16]

Taking this cue further, the athlete may be asked to perform the squat on a decline slant board (Figure 2).[17,18] The slant board encourages increased anterior knee translation, loading the patellar tendon further, as well as reducing the contribution of the gastroc-soleus complex to knee extension.[19] Additionally, single leg forward hops and single leg countermovement jumps provide a comparable high loading index and can be implemented as exercise progressions.[19] The location, staging and overall irritability of the tendinopathy should guide the clinician in exercise selection and cueing.

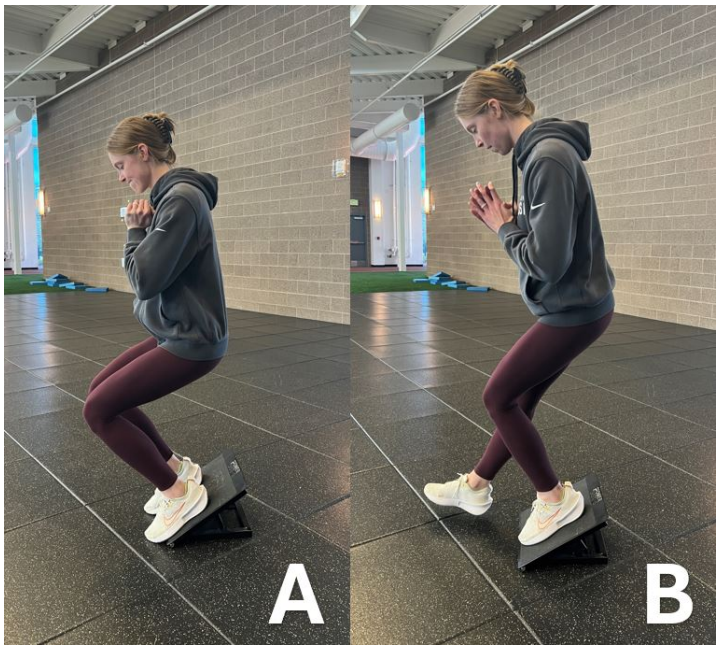

Figure 2. Decline squat variations on a slant board apply even more concentrated load to the patellar tendon.[17,18] A.) Weighted decline slant board squat. B.) Single leg slant board squat.

Forefoot Elevated (i.e.: Ankle Dorsiflexion) or Flat: Insertional versus Midportion Achilles Tendinopathy

Achilles tendinopathy can present in one of two categories, as defined by de Vois et al. in a recent multidisciplinary Achilles tendinopathy guideline publication.[11] Insertional Achilles tendinopathy is localized within the first 2 cm of the attachment of the Achilles tendon onto the calcaneus, while midportion Achilles tendinopathy is localized above this attachment.[11] The location of pain should influence exercise programming. For insertional Achilles tendinopathy, exercises should initially be performed on a flat surface[11], while midportion Achilles tendinopathy may be performed with the forefoot elevated (i.e., with the heels dipping off of the back of a step causing ankle dorsiflexion).[20] Compressive loads at the insertion of the Achilles tendon against the superior calcaneus occur when the forefoot is elevated, likely increasing pain levels acutely, and potentially exacerbating the tendinopathy due to direct pressure on the tendinopathic tissue.[21] A detailed clinical exam to discern the location of Achilles tendinopathy helps guide exercise selection.[11,21]

Load progression needs to be considered when designing an exercise program regardless of the location of the tendinopathy in the Achilles. Following the recommendations provided within the adaptation model proposed by Baxter et al, programming should begin with slow multi-joint movements progressing into dynamic single-leg movements to incrementally

increase Achilles tendon loading.[22] Examples of exercise with the lowest load index include seated heel raises whereas higher load index exercises would include standing heel raises, lunges, step-ups, and step-downs.[22] For progressions into plyometric exercises, which will have a higher load index compared to the previously referenced exercises due to increased peak loading and loading impulse, exercise dosing should first include exercises such as countermovement jumps, hopping, drop jumps and single leg heel raises before proceeding into single-leg variations of the same plyometric movements, including multidirectional hops, which load the Achilles tendon the most.[22]

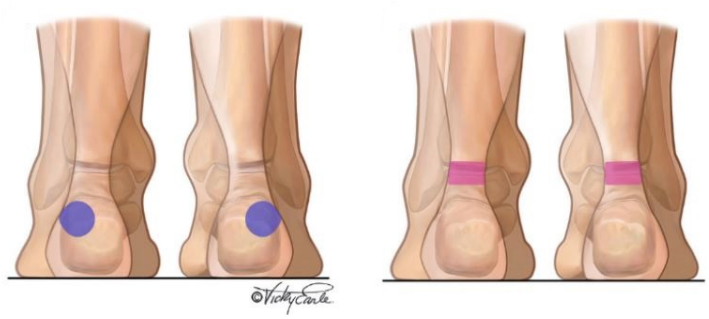

Figure 3. A typical pain presentation associated with insertional Achilles tendinopathy (feft). A typical pain presentation for midportion Achilles tendinopathy (right). Illustration with permission from Barça Innovation Hub 2021 Tendon Guide.[23]

The Influence of Hip Flexion Angles with Proximal Hamstring Tendinopathy

Injury to the hamstring musculature can occur in several locations (proximal, myotendinous junction, distal). Proximal hamstring tendinopathy typically affects the semimembranosus tendon.[24] High degrees of hip flexion may result in provocative tensile and compressive loads at the tendon insertion onto the ischial tuberosity.[25,26] Thus, hip flexion angles should be a major consideration depending on the stage and irritability of proximal hamstring tendinopathy. Acutely, hamstring loading should be performed with minimal hip flexion.[27] Sub-acutely, progressive hip flexion should be introduced as tolerance allows.[27] Late-stage rehabilitation should include plyometric programming[27] that involves high degrees of hip flexion.[28]

The Role of Impingement Mechanisms in Rotator Cuff Tendinopathy

Theories of mechanical impingement at the shoulder have existed for decades, claiming that pathologic contact between the undersurface of the acromion and the rotator cuff tendons can influence the presentation of tendinopathy. However, this model has been contested as the classic definitions of internal and external impingement do not account for a variety of

mechanisms and factors at play that may contribute to symptom presentation.[29] External impingement, or primary impingement, occurs due to changes that narrow the subacromial space, including an increase in the volume of subacromial tissues or anatomical abnormality such as a hooked (class III) acromion.[29,30] Internal - or secondary impingement - results from altered centering of the humeral head within the joint space and results in abnormal displacement during rest and overhead motion. Weakness in the rotator cuff, trapezius and serratus anterior muscles have been identified as contributors to internal impingement due to altered mechanics of the humeral head and scapula both at rest and during motion.[29,30] Additionally, functional adaptation to life or sport demands, including humeral retroversion, also influences the occurrence of pathologic internal impingement.[31] It is now thought that, given the complex interaction of multiple factors, shoulder impingement and rotator cuff tendinopathy potentially reinforce mechanisms of each condition and are not to be assumed to contribute in isolation.[32, 33]

As compared to the previous three sections (patellar, Achilles and proximal hamstring tendons), there are less movement considerations in relation to exercise selection for the rehabilitation of rotator cuff tendinopathy. Anecdotally, if programming for posterior cuff exercises, often in acute or subacute phases of tendinopathy, a small bolster can be placed between the elbow and body to slightly abduct the elbow, reducing supraspinatus tendon tension and irritability.[34]

There are a considerable number of publications that look at dynamic electromyography in the shoulder to help guide exercise selection; however, there is no direct relationship established between electromyography and actual tension in the correlated musculotendinous structures.[35] In general, electromyography signal increases in the supraspinatus, infraspinatus and teres minor as the shoulder reaches higher levels of horizontal abduction (90 degrees or more).[35] The level of shoulder abduction should be modified depending on staging and irritability of the rotator cuff tendinopathy.

References

1. Dunn SL, Olmedo ML. Mechanotransduction: Relevance to Physical Therapist Practice—Understanding Our Ability to Affect Genetic Expression Through Mechanical Forces. Phys Ther. 2016;96(5):712-721.
2. Arnoczky SP, Tian T, Lavagnino M, Gardner K, Schuler P, Morse P. Activation of stress-activated protein kinases (SAPK) in tendon cells following cyclic strain: the effects of strain frequency, strain magnitude, and cytosolic calcium. J Orthop Res. 2002;20(5):947-952.
3. Sun YL, Thoreson AR, Cha SS, Zhao C, An KN, Amadio PC. Temporal response of canine flexor tendon to limb suspension. J Appl Physiol. 2010;109(6):1762-1768.
4. Langberg H, Skovgaard D, Karamouzis M, Bülow J, Kjaer M. Metabolism and inflammatory mediators in the peritendinous space measured by microdialysis during intermittent isometric exercise in humans. J Physiol. 1999;515 (Pt 3)(Pt 3):919-927.
5. Irby A, Gutierrez J, Chamberlin C, Thomas SJ, Rosen AB. Clinical management of tendinopathy: A systematic review of systematic reviews evaluating the effectiveness of tendinopathy treatments. Scand J Med Sci Sports. 2020;30(10):1810-1826.
6. Kingma JJ, de Knikker R, Wittink HM, Takken T. Eccentric overload training in patients with chronic Achilles tendinopathy: a systematic review. Br J Sports Med. 2007;41(6):e3.
7. Murphy M, Travers M, Gibson W. Is heavy eccentric calf training superior to wait-and-see, sham rehabilitation, traditional physiotherapy and other exercise interventions for pain and function in mid-portion Achilles tendinopathy? Syst Rev. 2018;7(1):58.
8. Sussmilch-Leitch SP, Collins NJ, Bialocerkowski AE, Warden SJ, Crossley KM. Physical therapies for Achilles tendinopathy: systematic review and meta-analysis. J Foot Ankle Res. 2012;5(1):15.
9. Vander Doelen T, Jelley W. Non-surgical treatment of patellar tendinopathy: A systematic review of randomized controlled trials. J Sci Med Sport. 2020;23(2):118-124.
10. Murphy MC, Travers MJ, Chivers P, et al. Efficacy of heavy eccentric calf training for treating mid-portion Achilles tendinopathy: a systematic review and meta-analysis. Br J Sports Med. 2019;53(17):1070-1077.
11. de Vos RJ, van der Vlist AC, Zwerver J, et al. Dutch multidisciplinary guideline on Achilles tendinopathy. Br J Sports Med. 2021;55(20):1125-1134.
12. Couppé C, Svensson RB, Silbernagel KG, Langberg H, Magnusson SP. Eccentric or Concentric Exercises for the Treatment of Tendinopathies? J Orthop Sports Phys Ther. 2015;45(11):853-863.
13. 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. 2009;43(6):409-416.

14. Schoenfeld BJ. Squatting kinematics and kinetics and their application to exercise performance. J Strength Cond Res. 2010;24(12):3497-3506.
15. Fry AC, Smith JC, Schilling BK. Effect of knee position on hip and knee torques during the barbell squat. J Strength Cond Res. 2003;17(4):629-633.
16. Basas C, Ito N, Gravare Silbernagel K, Reyes-Gil F, Basas A. The Basas Spanish Squat: Superimposition of electrical stimulation to optimizie patellar tendon strain: a case series. Int J Sports Phys Ther. 2023;18(6): 1299-1307.
17. Malliaras P, Cook J, Purdam C, Rio E. Patellar Tendinopathy: Clinical Diagnosis, Load Management, and Advice for Challenging Case Presentations. J Orthop Sports Phys Ther. 2015;45(11):887-898.
18. Reinking MF. Current concepts in the treatment of patellar tendinopathy. Int J Sports Phys Ther. 2016;11(6):854-866.
19. Scattone Silva R, Song K, Hullfish T, Sprague A, Silbernagel K, Baxter J. Patellar tendon load progression during rehabilitation exercises: implications for the treatment of patellar tendon injuries. Med Sci Sports Exerc. 2024; 56(3):545-552.
20. Silbernagel KG, Crossley KM. A Proposed Return-to-Sport Program for Patients With Midportion Achilles Tendinopathy: Rationale and Implementation. J Orthop Sports Phys Ther. 2015;45(11):876-886.
21. Cook JL, Stasinopoulos D, Brismée JM. Insertional and mid-substance Achilles tendinopathies: eccentric training is not for everyone - updated evidence of non-surgical management. J Man Manip Ther. 2018;26(3):119-122.
22. Baxter J, Corrigan P, Hullfish T, O'Rourke P, Silbernagel K. Exercise progression to incrementally load the achilles tendon. Medicine and Science in Sports and Exercise. 2021;53(1), 124-130.
23. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
24. Askling C, Saartok T, Thorstensson A. Type of acute hamstring strain affects flexibility, strength, and time to return to pre-injury level. Br J Sports Med. 2006;40(1):40-44.
25. Docking S, Samiric T, Scase E, Purdam C, Cook J. Relationship between compressive loading and ECM changes in tendons. Muscles Ligaments Tendons J. 2013;3(1):7-11.
26. Soslowsky LJ, Thomopoulos S, Esmail A, et al. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. Ann Biomed Eng. 2002;30(8):1057-1063.
27. Goom TSH, Malliaras P, Reiman MP, Purdam CR. Proximal Hamstring Tendinopathy: Clinical Aspects of Assessment and Management. J Orthop Sports Phys Ther. 2016;46(6):483-493.

28. Schmitt B, Tim T, McHugh M. Hamstring injury rehabilitation and prevention of reinjury using lengthened state eccentric training: a new concept. Int J Sports Phys Ther. 2012;7(3):333-341.
29. Garving C, Jakob S, Bauer I, Nadjar R, Brunner U. Impingement syndrome of the shoulder. Deutsches Ärzteblatt International. 2017; 114(45), 765-776.
30. Cools A, Cambier D, Witvrouw E. Screening the athlete's shoulder for impingement symptoms: a clinical reasoning algorithm for early detection of shoulder pathology. Br J Sports Med. 2008; 42(8):628-635.
31. Ishikawa H, Kurokawa D, Muraki T, Nagamoto H, Takahashi H, Yamamoto N, Izumi S. Increased external rotation related to the soft tissues is associated with pathologic internal impingement in high-school baseball players. Journal of Shoulder and Elbow Surgery. 2022; 31(9),1823-1830.
32. Lewis J. Rotator cuff tendinopathy. Br J Sports Med. 2009;43:236–241.
33. Lewis J, McCreesh K, Roy J, Ginn, K. Rotator cuff tendinopathy: navigating the diagnosis-management conundrum. J Orthop Sports Phys Ther. 2015;45(11), 923-937.
34. Williams GR, Kelley M. Management of rotator cuff and impingement injuries in the athlete. J Athl Train. 2000;35(3):300-315.
35. Edwards PK, Ebert JR, Littlewood C, Ackland T, Wang A. A Systematic Review of Electromyography Studies in Normal Shoulders to Inform Postoperative Rehabilitation Following Rotator Cuff Repair. J Orthop Sports Phys Ther. 2017;47(12):931-944.

Nutrition Strategies for Tendon Health

Take Home Points

- Current research has yet to establish a direct link between nutritional interventions and prevention of tendinopathy. However, this does not preclude the potential benefits of targeted nutrition in strengthening tendons and reducing the risk of tendon injuries. Continued investigation is needed to better understand the relationship between dietary strategies and tendon health, particularly in the context of injury prevention.
- Suboptimal nutritional status may contribute to the risk of soft tissue injuries like tendinopathy. Dietary interventions targeting collagen synthesis may enhance tendon health and potentially prevent injuries.
- Protein is crucial for muscle recovery and tendon function, with different protein needs depending on activity level, body size, and goals. Consuming protein-rich foods post-exercise aids in muscle remodeling, while specific amino acids like leucine along with Vitamin C play vital roles in collagen synthesis, essential for tendon health.
- Adequate hydration is essential for tendon health; athletes should monitor hydration status and fluid intake before, during, and after activity. Additionally, limited evidence suggests that a moderate amount of alcohol consumption is correlated with Achilles tendinopathy.

While research on nutrition interventions for preventing soft tissue injuries is still in its early stages,[1,2] there is some evidence that suboptimal nutritional status is a risk factor for potential injuries, including tendinopathy.[3] Since diet plays a significant role in the homeostasis of all tissues, inadequate dietary practices are acknowledged as a potential factor contributing to the development of tendinopathy.[3] Dietary interventions may be able to enhance tendon health by supporting collagen synthesis [3] and provide the energy necessary for the mental and physical demands of proper movement.

Collagen is the structural backbone of intramuscular connective tissue, tendons, and ligaments, directly impacting their structural integrity and functional characteristics. The amount of collagen and the density of crosslinks within it significantly influences the mechanical properties of soft tissues. Most structural changes to tendons occur during maturation, and minimal additional alterations occur during the course of healthy aging,[4] although some work has demonstrated decreased collagen content in old (67 ± 3 years) versus young (27 ± 2 years) tendons.[5] Regardless, it is known that the accumulation of collagen progressively fortifies the tendon structure, heightening its ability to withstand injury.[2] While evidence is lacking, nutritional strategies may be able to assist in collagen synthesis,[6] offering opportunities to withstand mechanical stress[3] and expedite return to play.[2]

To mitigate the risk of soft tissue injury, the top nutritional priorities are meeting total energy intake, protein, and essential nutrient needs.[7] Although the specific energy needs of individuals vary considerably, athletes are recommended to habitually consume a well-balanced diet to support training, prevent illness, and aid in recovery from both training and competition. Athletes should ensure a diverse dietary intake, incorporating foods abundant in vitamins, minerals, and essential amino acids to support protein and collagen synthesis. Athlete Plates, designed in collaboration between the United States Olympic and Paralympic Committee and the University of Colorado at Colorado Springs, serve as a visual aid and a practical tool to illustrate nutritional portions aligned with energy expenditure across different training days (e.g., an easy day, moderate day, and a hard training day). [8]

Protein aids in muscle recovery by providing the building blocks (amino acids) necessary for repairing and rebuilding muscle tissue damaged during exercise. Eating a variety of lean protein sources such as poultry, fish, beef, beans, soy, milk, and legumes provides the body with a variety of amino acids needed to optimize muscle and tendon function.[2] Total protein needs differ individually based on body size, activity level, sport type, injury status, and goals (Table 1).

Table 1. Protein needs for various athletes with examples.

Activity Level	Protein Needs (g/kg/day)	Example Athlete 1 (150lbs, 68kg) Needs (g)	Example Athlete 1 (180lbs, 82kg) Needs (g)
Average, non-athlete	0.8 - 1.0	54 - 68	66 - 82
Endurance Athlete	1.2 - 1.4	82 - 95	102 - 115
Power Athlete	1.5 - 1.8	102 - 116	123 - 140
Injured or Weight Loss Athlete	1.8 - 2.2	122 - 150	148 - 180

Protein consumption within 30 to 60 minutes after exercise maximizes the muscle remodeling process.[9] Specific to tendon health, it has been demonstrated that consuming a whey protein supplement (19.5g high-leucine whey protein and 19.5g carbohydrate) on all training days for 12 weeks was associated with a potential increase in tendon hypertrophy.[6] The ideal dose of leucine is 2-3g per meal and snack, along with a variety of amino acids to maximize muscle protein synthesis,[10] which can be accomplished by the consumption of 5 oz of chicken or 6 oz of fish. Animal models have suggested that a leucine-rich whey protein supplement can activate mTORC1 within sinews, leading to increased collagen production,[11] implying that integrating leucine-rich whey protein foods into a training routine could provide advantages extending beyond muscle development.

Collagen molecules consist of non-essential amino acids, particularly glycine, proline, and hydroxyproline. Collagen-rich foods include bone broth, gelatin (which is made from the tendons and ligaments of cows), beef or chicken with skin or around bones, and fish with edible skin and bone. [1,3] While muscles benefit from protein reinforcement post-exercise, tendons, with their limited blood flow, rely on bulk fluid flow for nutrient delivery that is upregulated during activity. [2,3] In addition to specific amino acids,[12] Vitamin C is a vital cofactor for an enzyme essential in the synthesis and cross-linking of collagen and thus should be prioritized for tendon health. Vitamin C-rich foods include oranges, lemons, limes, grapefruits, berries, kiwi, pineapple, mango, papaya, bell peppers, broccoli, brussels sprouts, spinach, kale, tomatoes, potatoes, and cantaloupe.[13] It has been demonstrated that consuming 15 grams of vitamin C-enriched collagen 60 minutes before activity significantly increases collagen synthesis[14] and will be discussed further in the Nutrition for Injured Tendons section.

Carbohydrates should constitute a relatively large percentage of an athlete's diet, as represented by a percentage of total calories consumed, and should be adjusted based on body mass and energy expenditure (Table 2). Adequate carbohydrate intake ensures sufficient energy to support tendon remodeling, repair, and maintenance processes. While it is widely understood that carbohydrates are an essential nutrient for sports performance and muscle recovery, tendon glucose uptake also increases during exercise.[15] Carbohydrate food sources include pasta, rice, cereals, bread, oats, crackers, fruits, starchy vegetables, and sports performance products like bars, chews, and sports drinks.

Table 2. Carbohydrate needs for various athletes with examples.

Training Type	Carbohydrate Recommendations (g/kg/day)	Example Athlete 1 (150lbs, 68kg) Needs (g)	Example Athlete 1 (180lbs, 82kg) Needs (g)
Low-intensity or skill-based activities	3 - 5	204 - 340	246 - 410
Moderate (~1 hour/day)	5 - 7	340 - 476	410 - 574
Endurance (1-3 hours/day)	6 - 10	408 - 680	492 - 820
Ultra-endurance (≥4 hours/day)	8 - 12	544 - 816	656 - 984

A well-rounded diet includes essential fatty acids, such as monounsaturated fatty acids (MUFAs) and polyunsaturated fatty acids (PUFAs), including both omega-3 and omega-6 fatty acids. Omega-3 fatty acids are known for their anti-inflammatory properties[16] and thus may contribute to reducing exercise-induced muscle damage and soreness,[17,18] although this is not necessarily a consistent effect.[19] To integrate essential fatty acids into a healthy diet, athletes should prioritize omega-3-rich foods like salmon, mackerel, sardines, anchovies, tuna, walnuts, and flax seeds.[20] However, one should be cognizant of moderating the intake of MUFA, PUFA, and saturated fats as part of a balanced diet.

Tendons are composed of over 60% water and rely on bulk fluid flow for nutrient delivery.[3] Additionally, alongside crosslinked collagen, water contributes to the tendon's viscoelastic nature.[7] To ensure proper hydration to maximize tendon health (along with numerous other health and performance benefits), athletes should aim to maintain a normal state of hydration throughout the day. This includes that athletes meet daily water intake recommendations: 2.0 – 2.7 liters (70 – 90 ounces) per day for women and 2.5 – 3.7 liters (85 – 125 ounces) per day for men [21,22], though individual needs may vary, especially as training load, volume, and intensity vary, and with variations in training climate. Athletes can monitor their day-to-day hydration status by monitoring their first-morning urine color, body weight, and thirst perception.[23] If urine color is dark (e.g., it looks like apple juice), they feel thirsty, and/or their body weight is >1% less than the previous day, the athlete may be falling short of the aforementioned guidelines. It is also important for athletes to maintain their hydration status during and after an activity. Consuming fluids during activity with the goal of minimizing body mass losses via sweat and replacing remaining deficits following exercise will optimize one's fluid intake strategy during exercise.[24] To know how much fluid one should consume after activity, athletes should weigh themselves before and after activity (preferably nude or in minimal clothing), and any difference in body weight is the amount of fluid that needs to be consumed (for every 1 lb. of body weight loss, 16 – 24 ounces of fluid are needed to replace that loss). [23, 24]

There is limited research on the impact of alcohol on tendon health, with discrepancies in study designs, participant groups, and definitions of alcohol consumption levels, restricting the ability to make direct comparisons across studies. However, animal models have demonstrated that ethanol can impair tendon collagen organization and delay healing.[25] In humans, excessive alcohol consumption (more than 13 drinks for men and more than 6 drinks for women per week), has been associated with a significantly greater risk and severity of rotator cuff tears,[26] while moderate weekly alcohol consumption (i.e., 7 to 13 drinks for men and 4 to 6 drinks for women per week) has been associated with a small increase in the risk of Achilles tendinopathy.[27] Alcohol consumption also negatively affects hydration status, potentially suppressing the anabolic response in skeletal muscle and impairing recovery and adaptation to training, thereby increasing the risk of injury.[28] Lastly, efforts to minimize alcohol intake will help maintain overall fluid balance since alcohol inhibits an essential hormone (arginine vasopressin) that controls body water balance within the body.[29,30]

Nutrition assessments completed by a registered dietitian (RD) are important for optimizing nutrient intake for performance, recovery, and injury prevention and monitoring risk for deficiencies. Much of the current understanding of nutrition and tendon health is extrapolated from muscle-focused literature, which suggests potential indirect benefits for tendons. However, the direct effects of nutritional interventions on tendon tissue remain less understood, highlighting the need for more tendon-specific research.

References

1. Curtis L. Nutritional research may be useful in treating tendon injuries. Nutrition. 2016;32(6):617–9.
2. Baar K. Minimizing injury and maximizing return to play: Lessons from engineered ligaments. Sports Med. 2017;47:5–11.
3. Hijlkema A, Roozenboom C, Mensink M, Zwerver J. The impact of nutrition on tendon health and tendinopathy: a systematic review. J Int Soc Sports Nutr. 2022;19(1):474–504.
4. Svensson RB, Heinemeier KM, Couppé C, Kjaer M, Magnusson SP. Effect of aging and exercise on the tendon. J Appl Physiol. 2016;121(6):1353–62.
5. Couppe C, Hansen P, Kongsgaard M, Kovanen V, Suetta C, Aagaard P, et al. Mechanical properties and collagen cross-linking of the patellar tendon in old and young men. J Appl Physiol. 2009;107(3):880–6.
6. Farup J, Rahbek S, Vendelbo M, Matzon A, Hindhede J, Bejder A, et al. Whey protein hydrolysate augments tendon and muscle hypertrophy independent of resistance exercise contraction mode. Scand J Med Sci Sports. 2014;24(5):788–98.
7. Tipton KD. Nutritional support for exercise-induced injuries. Sports Med. 2015;45(Suppl 1):93–104.
8. United States Olympic Committee Sport Dietitians and the University of Colorado (UCCS) Sport Nutrition Graduate Program. Athlete Plates. USOPC.org. Accessed January 14, 2025. https://www.usopc.org/nutrition
9. Bonilla DA, Pérez-Idárraga A, Odriozola-Martínez A, Kreider RB. The 4r's framework of nutritional strategies for post-exercise recovery: A review with emphasis on new generation of carbohydrates. Int J Environ Res Public Health. 2021;18(1):103.
10. Reidy PT, Rasmussen BB. Role of ingested amino acids and protein in the promotion of resistance exercise–induced muscle protein anabolism. J Nutr. 2016;146(2):155–83.
11. Barbosa AWC, Benevides GP, Alferes LMT, Salomão EM, Gomes-Marcondes MCC, Gomes L. A leucine-rich diet and exercise affect the biomechanical characteristics of the digital flexor tendon in rats after nutritional recovery. Amino Acids. 2012;42:329–36.
12. Paxton JZ, Grover LM, Baar K. Engineering an in vitro model of a functional ligament from bone to bone. Tissue Eng Part A. 2010;16(11):3515–25.
13. National Institute of Health - Office of Dietary Supplements. Vitamin C - Fact Sheet for Health Professionals. [cited 2024 Mar 25]. Office of Dietary Supplements - Vitamin C. Available from: https://ods.od.nih.gov/factsheets/VitaminC-HealthProfessional/
14. Shaw G, Lee-Barthel A, Ross ML, Wang B, Baar K. Vitamin C–enriched gelatin supplementation before intermittent activity augments collagen synthesis. Am J Clin Nutr. 2017;105(1):136–43.

15. Bojsen-Møller J, Kalliokoski KK, Seppanen M, Kjaer M, Magnusson S. Low-intensity tensile loading increases intratendinous glucose uptake in the Achilles tendon. J Appl Physiol. 2006;101(1):196–201.
16. Calder PC. Omega-3 fatty acids and inflammatory processes. Nutrients. 2010;2(3):355–74.
17. Kyriakidou Y, Wood C, Ferrier C, Dolci A, Elliott B. The effect of Omega-3 polyunsaturated fatty acid supplementation on exercise-induced muscle damage. J Int Soc Sports Nutr. 2021;18:1–11.
18. Jouris KB, McDaniel JL, Weiss EP. The effect of omega-3 fatty acid supplementation on the inflammatory response to eccentric strength exercise. J Sports Sci Med. 2011;10(3):432.
19. Visconti LM, Cotter JA, Schick EE, Daniels N, Viray FE, Purcell CA, et al. Impact of varying doses of omega-3 supplementation on muscle damage and recovery after eccentric resistance exercise. Metab Open. 2021;12:100133.
20. National Institute of Health - Office of Dietary Supplements. Omega-3 Fatty Acids - Fact Sheet for Health Professionals. [cited 2024 Mar 26]. Office of Dietary Supplements - Omega-3 Fatty Acids. Available from: https://ods.od.nih.gov/factsheets/Omega3FattyAcids-Consumer/
21. EFSA Panel on Dietetic Products, Nutrition, and Allergies (NDA). Scientific Opinion on Dietary Reference Values for Water. EFSA J. 2010;8(3):1459.
22. Standing Committee on the Scientific Evaluation of Dietary Reference Intakes, Panel on Dietary Reference Intakes for Electrolytes, Water. Dietary reference intakes for water, potassium, sodium, chloride, and sulfate. National Academies Press; 2005.
23. Cheuvront SN, Sawka MN. Hydration assessment of athletes. Sports Sci Exch. 2005;18(2):1–6.
24. McDermott BP, Anderson SA, Armstrong LE, Casa DJ, Cheuvront SN, Cooper L, et al. National athletic trainers' association position statement: fluid replacement for the physically active. J Athl Train. 2017;52(9):877–95.
25. Hapa O, Çakıcı H, Gideroğlu K, Özturan K, Kükner A, Buğdaycı G. The effect of ethanol intake on tendon healing: a histological and biomechanical study in a rat model. Arch Orthop Trauma Surg. 2009;129:1721–6.
26. Passaretti D, Candela V, Venditto T, Giannicola G, Gumina S. Association between alcohol consumption and rotator cuff tear. Acta Orthop. 2016;87(2):165–8.
27. Owens BD, Wolf JM, Seelig AD, Jacobson IG, Boyko EJ, Smith B, et al. Risk factors for lower extremity tendinopathies in military personnel. Orthop J Sports Med. 2013;1(1):2325967113492707.
28. Medina D, Lizarraga A, Drobnick F. Injury prevention and nutrition in football. Sports Sci Exch. 2014;27(132):1–5.
29. Shirreffs SM, Maughan RJ. Restoration of fluid balance after exercise-induced dehydration: effects of alcohol consumption. J Appl Physiol. 1997;83(4):1152–8.

30. Wijnen AH, Steennis J, Catoire M, Wardenaar FC, Mensink M. Post-exercise rehydration: effect of consumption of beer with varying alcohol content on fluid balance after mild dehydration. Front Nutr. 2016;3:45.
31. van der Vlist AC, Breda SJ, Oei EHG, Verhaar JAN, de Vos RJ. Clinical risk factors for Achilles tendinopathy: a systematic review. Br J Sports Med. 2019;53(21):1352-1361. doi:10.1136/bjsports-2018-099991.

The Injured Tendon

An Overview of the Injured Tendon

Take Home Points

- The pathogenesis of tendinopathy is multifactorial and complex. The onset of tendinopathy appears to be related to repetitive tendon overload, leading to structural injury of the collagen fibrils.[1]
- The initial structural alterations associated with tendon damage are likely asymptomatic. Over time, the accumulation of matrix damage and chemical mediators activate nociceptors leading to clinical manifestation.[1]
- Many models of tendinopathy have been proposed since the 1970s. Modern day models focus more on a continuum of tendon pathology,[2,3] while older models focused more on an inflammatory model. The inflammatory model is largely disputed.[4,5]
- Tendinopathy management models have become more holistic and somewhat forgiving for the clinician. While continuum models are helpful conceptually, the associated histology makes classification of the stage of tendinopathy difficult. Management models should remain patient-centered and personalized.[1]

Tendinopathy is defined as persistent tendon pain and loss of function related to mechanical loading.[6,7] During bouts of unaccommodated exercise or training, repetitive contraction of the muscle precipitates persistent micro-trauma of tendons that often lack the appropriate recovery time due to slow collagen turnover.[7,8] These morphological changes result in a tendon that is less capable of sustaining repeated tensile load and, unfortunately, due to general hypocellularity and hypovascularity, the healing ability of the tendon is fairly inefficient.[9]

Load (i.e. active exercise, rehabilitation) is thought to be one of the most influential variables in the development and management of tendinopathy;[10] however, other intrinsic factors like genes, age, circulating and local cytokine production, sex, biomechanics and body composition are often at play.[2] In the last several decades of rehabilitation, tendinopathies have generally been approached the same regardless of these pathoaetiological considerations. Applying the same rehabilitative interventions, regardless of these considerations, is likely to be ineffective in many cases. Unfortunately, injured tendons heal slowly, often resulting in impaired structural integrity and mechanical strength needed for the demands of sport.[11] As a result, both athletes and coaching staff are burdened by decreased athlete availability and performance.

Microscopically, healthy tendons are predominantly composed of parallel, closely packed collagen fibers and cells within a well-ordered extracellular matrix.[7,11] Type I collagen makes up about 65% to 80% of the tendon dry mass.[11] Normally, these Type I collagen fibers are flexible, strong, and resistant to repetitive damage. Over time, repetitive tensile loading can lead to an accumulation of microdamage. As a result, scattered inefficient vascular in-growth, including ineffective capillaries, contribute to vascular compromise and local tissue hypoxia.[12] Table 1 (from *Khan & Brukner, 2017)* summarizes the maladaptive cellular responses that occur when the tendon is exposed to excessive loading.[13]

Table 1: Five elements of normal tendon compared with the characteristic elements of end-stage tendon overuse injury[13] (adapted from Khan & Brukner 2017, with permission from original authors)

Tendon Element	*Normal Tendon*	*Changes that occur in response to excessive tendon loading*
Cells – tenocytes	Tendon cells are spindle-shaped, and nuclei cluster in longitudinal chains on microscopy	Tissue has proliferation of cells with abnormally rounded nuclei and areas with fewer than normal cell numbers
Ground substance or "matrix" proteins	The ground substance in the matrix is minimal and is not visible when stained for light microscopic viewing	Increased amount of ground substance/matrix proteins which stain and are visible under light microscopy
Collagen	Linear and tightly bundled and has a characteristic crimp under polarized light	Disrupted – both longitudinally and in its bundles
Nerves	Minimal neurotendinous nerves, some innervation of connective tissue in and around the tendon	Abnormal ingrowth of nerves and a preponderance of neuropeptides
Vessels	Minimal vascularity when examined histologically or by using ultrasound	Prominent vessels histologically or using ultrasound

In the late 1970s,[4,14–16] all tendon injuries were typically considered to be inflammatory in nature (i.e. tendonitis). Decades later, histopathologic literature has evolved and chronic tendinopathies have been observed to be absent of cellular inflammation.[7,17,18,] Typical pathological changes that occur in tendinopathy include reduced numbers and rounding of fibroblasts, an increase in the content of proteoglycans, glycosaminoglycans and water, hypervascularization, nerve ingrowth and disorganized fibrils.[7] Very little is actually known

regarding the actual mechanisms of how health tendon tissue accumulates damage.[19] Tendon damage may occur from acute tearing (cutting, laceration), oxidative damage,[20] accumulation of microtears,[21–25] or the production of aberrant matrix within the tendon such as ectopic calcification.[26,27]

Numerous theories on the pathogenesis of tendinopathy exists in the literature.[1] Over time, these have developed with the field leaning toward modern continuum models, although all are generally helpful in correlating basic tendon science to clinical findings and evaluation.[1] These theories include the *mechanical theory*[28] (one of the first theories proposed in the late 70s), the *inflammation model* (which has been disputed by most[4,5]), the *apoptosis theory,*[29,30] *vascular / neurogenic theory*[31] and lastly the *continuum model*.[2,3]

Several tendinopathy continuum models have been proposed in the last 1 to 2 decades including Cook's continuum model of tendinopathy,[2] subsequent adaptations by other researchers[32] and revisions by Cook et al.[3] These models encouraged clinicians to monitor and prescribe graded load based on the staging of tendinopathy. These were monumental developments at a time where many in the field prescribed outdated interventions like complete rest and anti-inflammatory medications.[1] While helpful conceptually, these models often encourage the rehabilitative clinician to speculate on the stage of tendinopathy to guide treatment (*normal tendon, reactive tendon, tendon disrepair, degenerative tendon*).[2,3] Without histological confirmation, clinicians were typically left to make an educated guess based on patient reports and objective exam. A newer management model **(Figure 1)**, published by Millar et al. in 2021, focuses less on tendinopathy staging and more on a holistic treatment model.[1] This model is explored more in the "General Tendinopathy Management Guidelines" section.

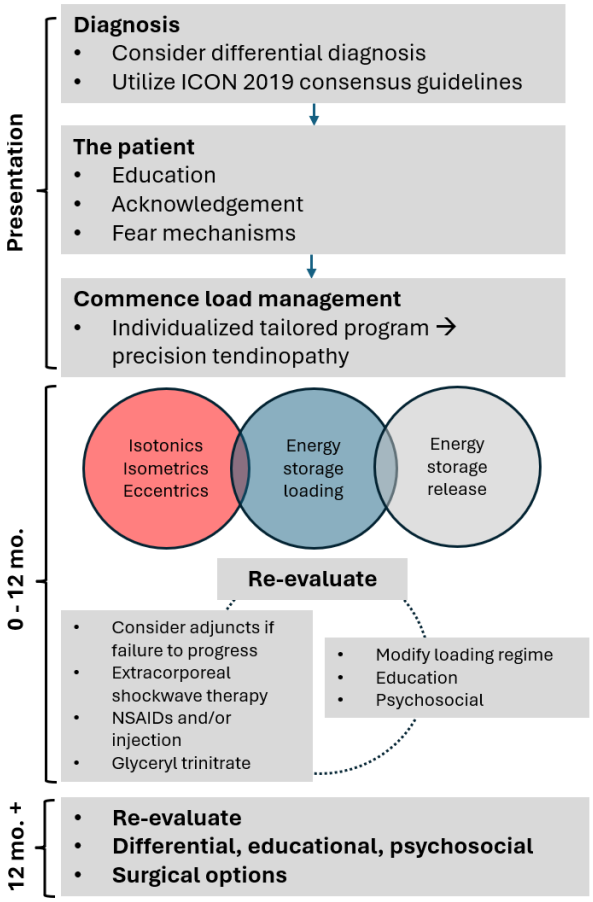

Figure 1: Adapted from Millar et al, 2021. This model generalizes timing familiar to the clinician including presentation, 0 to 12 months, and 12+ months. It takes into account general loading strategies, frequent re-evaluations, and adjunct treatments should progress when individualized programming begins to stall.[1] The clinician is not required to stage the tendinopathy or map where treatment should begin.

Snedeker and Foolen published a tendon injury model that theorizes by the time an individual encounters clinical symptoms associated with tendinopathy there has already been an underlying, long-term, sub-symptom accumulation of tissue damage.[19] They named this onset of symptoms the "metabolic tipping point."[19] This point is theorized to be when the accumulation of intrinsic tendon damage exceeds the metabolic demands and nutrient supply of the normally avascular core.[33] The extrinsic tendon has a faster metabolism and manages daily remodeling, while the intrinsic core is slower and adapts to loading over time.[19] There is a close interplay theorized between the intrinsic compartment and extrinsic compartment of a tendon.[34–36] When this tipping point occurs, it is theorized that the extrinsic tendon is recruited by the

intrinsic tendon to participate in organ / tissue remodeling,[19] thus the neovascularization and innervation often seen associated with chronic tendon disease.[37]

References

1. Millar NL, Silbernagel KG, Thorborg K, et al. Tendinopathy. Nat Rev Dis Primers. 2021;7(1):1.
2. 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. 2009;43(6):409-416.
3. Cook JL, Rio E, Purdam CR, Docking SI. Revisiting the continuum model of tendon pathology: what is its merit in clinical practice and research? Br J Sports Med. 2016;50(19):1187-1191.
4. Puddu G, Ippolito E, Postacchini F. A classification of Achilles tendon disease. Am J Sports Med. 1976;4(4):145-150.
5. Khan KM, Cook JL, Bonar F, Harcourt P, Astrom M. Histopathology of common tendinopathies. Update and implications for clinical management. Sports Med. 1999;27(6):393-408.
6. Scott A, Squier K, Alfredson H, et al. ICON 2019: International scientific tendinopathy symposium consensus: Clinical terminology. Br J Sports Med. 2020;54(5):260-262.
7. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. Nat Rev Rheumatol. 2010;6(5):262-268.
8. Peace KAL, Lee JC, Healy J. Imaging the infrapatellar tendon in the elite athlete. Clin Radiol. 2006;61(7):570-578.
9. Benjamin M, Ralphs JR. Tendons and ligaments: An Overview. Histol Histopathol. 1997;12:1135-1144.
10. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
11. Wu F, Nerlich M, Docheva D. Tendon injuries: Basic science and new repair proposals. EFORT Open Rev. 2017;2(7):332-342.
12. Järvinen M, Józsa L, Kannus P, Järvinen TL, Kvist M, Leadbetter W. Histopathological findings in chronic tendon disorders. Scand J Med Sci Sports. 1997;7(2):86-95.
13. Brukner P, Khan K, Hutchinson M. Brukner & Khan's Clinical Sports Medicine. Clinical Sports Medicine: Injuries. 2017;1.
14. Rocha e Silva M. A brief survey of the history of inflammation. Agents Actions. 1978;8(1-2):45-49.
15. Leadbetter W. An introduction to sports induced soft tissue inflammation. Sports Induced Inflammation; Clinical & amp; amp; Basic Science Concepts. Published online 1990. https://cir.nii.ac.jp/crid/1571980075660237568
16. Scott A, Khan KM, Cook JL, Duronio V. What is "inflammation"? Are we ready to move beyond Celsus? Br J Sports Med. 2004;38(3):248-249.

17. Rees JD, Stride M, Scott A. Tendons--time to revisit inflammation. Br J Sports Med. 2014;48(21):1553-1557.
18. Khan KM, Cook JL, Kannus P, Maffulli N, Bonar SF. Time to abandon the “tendinitis” myth. BMJ. 2002;324:626-627.
19. Snedeker JG, Foolen J. Tendon injury and repair - A perspective on the basic mechanisms of tendon disease and future clinical therapy. Acta Biomater. 2017;63:18-36.
20. Snedeker JG, Gautieri A. The role of collagen crosslinks in ageing and diabetes - the good, the bad, and the ugly. Muscles Ligaments Tendons J. 2019;04(03):303.
21. Shepherd JH, Screen HRC. Fatigue loading of tendon. Int J Exp Pathol. 2013;94(4):260-270.
22. Fung DT, Wang VM, Laudier DM, et al. Subrupture tendon fatigue damage. J Orthop Res. 2009;27(2):264-273.
23. Schechtman H, Bader DL. Fatigue damage of human tendons. J Biomech. 2002;35(3):347-353.
24. Schechtman H, Bader DL. In vitro fatigue of human tendons. J Biomech. 1997;30(8):829-835.
25. Wren TAL, Lindsey DP, Beaupré GS, Carter DR. Effects of creep and cyclic loading on the mechanical properties and failure of human Achilles tendons. Ann Biomed Eng. 2003;31(6):710-717.
26. Lui PPY, Chan LS, Cheuk YC, Lee YW, Chan KM. Expression of bone morphogenetic protein-2 in the chondrogenic and ossifying sites of calcific tendinopathy and traumatic tendon injury rat models. J Orthop Surg Res. 2009;4(1). doi:10.1186/1749-799x-4-27
27. Takeuchi E, Sugamoto K, Nakase T, et al. Localization and expression of osteopontin in the rotator cuff tendons in patients with calcifying tendinitis. Virchows Arch. 2001;438(6):612-617.
28. Burry HC. Pathogenesis of some traumatic and degenerative disorders of soft tissue. Aust N Z J Med. 1978;8 Suppl 1:163-167.
29. Yuan J, Murrell GAC, Trickett A, Wang MX. Involvement of cytochrome c release and caspase-3 activation in the oxidative stress-induced apoptosis in human tendon fibroblasts. Biochim Biophys Acta Mol Cell Res. 2003;1641(1):35-41.
30. Yuan J, Murrell GAC, Wei AQ, Wang MX. Apoptosis in rotator cuff tendonopathy. J Orthop Res. 2002;20(6):1372-1379.
31. Pufe T, Petersen WJ, Mentlein R, Tillmann BN. The role of vasculature and angiogenesis for the pathogenesis of degenerative tendons disease. Scand. J Med Sci Sports. 2005;15:211-222.
32. Lewis JS. Rotator cuff tendinopathy: a model for the continuum of pathology and related management. Br J Sports Med. 2010;44(13):918-923.
33. Ackermann PW, Hart DA. General overview and summary of concepts regarding tendon disease topics addressed related to metabolic disorders. Adv Exp Med Biol. 2016;920:293-298.

34. Liu CF, Aschbacher-Smith L, Barthelery NJ, Dyment N, Butler D, Wylie C. What we should know before using tissue engineering techniques to repair injured tendons: a developmental biology perspective. Tissue Eng Part B Rev. 2011;17(3):165-176.
35. Ackermann PW, Salo P, Hart DA. Tendon innervation. Adv Exp Med Biol. 2016;920:35-51.
36. Ackermann PW. Neuronal pathways in tendon healing and tendinopathy - update. Front Biosci. 2014;19(8):1251.
37. Rio E, Moseley L, Purdam C, et al. The pain of tendinopathy: physiological or pathophysiological? Sports Med. 2014;44(1):9-23.

Tendinopathy: Time to Return to Play in Elite Athletes

Take Home Points

- Return-to-play timelines are highly variable, with literature often citing return-to-play timelines anywhere between 3 months to >1 year. Unfortunately, such large timeline ranges are often unhelpful when educating athletes and coaching personnel.
- Rehabilitative literature generally suggests that longer intervention durations (>12 weeks) are typically more beneficial than shorter ones. However, symptoms may persist despite well-developed rehabilitation plans. Rehabilitation should continue even after the cessation of symptoms.
- Average return-to-play timelines in the literature should be interpreted with caution. Subject pools are often non-specific to elite athletes and generally include the public. Despite the paucity of literature permitting generalizations, the authors have opted to include reported timelines in this section.

Return-to-play timelines vary considerably following the onset of tendinopathy. Compared to typical soft tissue injuries, tendinopathies tend to follow the same healing phases: the inflammatory phase (1 to 7 days), the proliferative phase (7 to 21 days), and the remodeling phase (3 weeks to 1 year).[1] Fibroblasts synthesize type III collagen in the proliferative phase, which is gradually replaced by type I collagen from day 12-14, with subsequent and gradual increases in tensile strength.[2] Recovery of tendon injuries are slow due to low oxygen consumption[3] and slow synthesis of structural protein.[1]

Mechanotransduction, the process of translating mechanical stimuli into biological responses, is a major intervention used by clinicians to encourage tendon remodeling.[4] Mechanotransduction is skillfully applied during rehabilitation; studies generally include an intervention for a minimum of 12 weeks,[5] with return to sport occurring thereafter.[1,6] However, this is certainly not representative of all return-to-play timelines in tendinopathy. Rehabilitation can often extend deeply into the remodeling phase (3 weeks to 1 year) with elite athletes who, anecdotally, often struggle with recalcitrant symptoms at the highest demands of their sport (e.g., sprinting, change of direction, etc.).

Unfortunately, large non-specific return-to-play timeline windows are generally unhelpful when educating athletes and coaching personnel. To the knowledge of the authors, few high-quality studies, meta-analyses or systematic reviews on return-to-play timelines in tendinopathies currently exist in the literature. A full return to sport should be completed only

when symptoms are no longer provoked by exercise and the resolution of strength deficits has occurred.[7,8] Importantly, symptom resolution alone does not ensure readiness to return to play. Tendon structural alterations and functional deficits such as those previously mentioned should be considered, as these can persist well beyond symptoms. During the rehabilitation period, clinicians should use multiple strategies to gauge the athlete's progress and guide the modifications required until full sport participation. A gradual and controlled process that includes recovery days is key,[9] whereby clinicians may guide the gradual return based on, among the factors already discussed, the pain-monitoring model.[9,10] Thankfully, not all cases of tendinopathy result in lost player availability. A 2019 study found that 1 in every 3 cases of tendinopathy resulted in time loss,[8] indicating most athletes continue to play with tendinopathy.

Achilles Tendinopathy

In general, athletes with Achilles tendinopathy can be expected to return to sport anywhere between 6 weeks to 1 year after the initial onset of injury.[9] In a 2009 systematic review of nonoperative treatment for midportion tendinopathy,[11] 4 randomized clinical trials collectively showed a return to sport ranging from 10% to 86%.[12–14] A 2021 Dutch multidisciplinary guideline on Achilles tendinopathy suggests 85% of athletes return to sport; however, it is unknown if this is at the original performance level or fully asymptomatic.[15] The authors go as far to recommend that clinicians should inform athletes about the inability to provide a long-term prognosis, as prognostic factors for recovery have not yet been identified.[15] In the worst-case scenario, symptoms may persist for up to 10 years, with 23% to 37% reporting persistent symptoms despite treatment.[15]

Patellar Tendinopathy

Patellar tendinopathy is present in approximately 14% of all elite athletes at any given time, and 22% of elite athletes will have patellar tendon pain at some point in their athlete career.[16] The most common outcome associated with patellar tendinopathy is a loss of participation.[17] Return-to-play timelines are widely variable, with some reports of chronic impairment for an average duration of 19 months.[18] Other literature reports impairment up to 32 months in elite athletes.[19] Another study reported that 33% of patellar tendinopathy cases may struggle with time loss in sports due to symptoms for more than 6 months.[4]

A 2021 study following 56 NBA athletes with symptomatic patellar tendinopathy that was significant enough to be reported to the media found that 100% of athletes eventually returned to play following diagnosis. The mean (standard deviation) time between diagnosis and return to play was 60.7 ± 95.2 days. Twenty-two players missed fewer than 10 days, while 9 players only missed 1 to 2 days.[20] Athletes were able to return to play without any impact on career longevity.[20] Another prospective study performed by Gemignani et al. with 282

professional or semiprofessional athletes with patellar tendinopathy found return to play was faster in those who underwent conservative management than operative management.[21] Tendon involvement with musculoskeletal ultrasound could also be used to predict a return to play in conservatively managed athletes (grade 1 tendinopathy = 20 days, grade 2 tendinopathy = 40 days, grade 3 tendinopathy = 90 days, where grading was completed via sonographic evaluation of the relative injured area of the tendon).[21]

Proximal Hamstring Tendinopathy

The specific range or estimates for the time to return to play following recognition and diagnosis of proximal hamstring tendinopathy are not clearly established in the literature and are dependent on the severity of the condition. An expert opinion suggests that conservative treatment of proximal hamstring tendinopathy results in full recovery within one to three months.[22] A single study of 90 professional and competitive athletes indicated surgical intervention to treat proximal hamstring tendinopathy, with most athletes able to return to sport at the same level within 2 to 12 months.[23]

Conclusions

The scientific literature currently lacks the comprehensive data necessary to determine the time required to return to sport following the identification and diagnosis of tendinopathy. Further research is warranted in this area to permit the evaluation of various treatment and rehabilitation strategies against these expected timelines, which will depend on the etiology and severity of the specific tendinopathy.

References

1. Kader D, Saxena A, Movin T, Maffulli N. Achilles tendinopathy: some aspects of basic science and clinical management. Br J Sports Med. 2002;36(4):239-249.
2. LG J, P K. Human tendons: anatomy, physiology and pathology. Hum Kinet. Published online 1997.
3. Vailas A, Tipton C, Laughlin H, Tcheng T, Matthes R. Physical activity and hypophysectomy on the aerobic capacity of ligaments and tendons. J Appl Physiol. 1978;44(4):542-546.
4. Cook J, Khan K, Harcourt P, Grant M, Young D, Bonar S. A cross sectional study of 100 athletes with jumper's knee managed conservatively and surgically. The Victorian Institute of Sport Tendon Study Group. Br J Sports Med. 1997;31(4):332-336.
5. Bohm S, Mersmann F, Arampatzis A. Human tendon adaptation in response to mechanical loading: a systematic review and meta-analysis of exercise intervention studies on healthy adults. Sports Med-Open. 2015;1:1-18.
6. Paavola M, Kannus P, Järvinen TA, Khan K, Józsa L, Järvinen M. Achilles tendinopathy. JBJS. 2002;84(11):2062-2076.
7. Hägglund M, Waldén M, Ekstrand J. Lower reinjury rate with a coach-controlled rehabilitation program in amateur male soccer: a randomized controlled trial. Am J Sports Med. 2007;35(9):1433-1442.
8. Florit D, Pedret C, Casals M, Malliaras P, Sugimoto D, Rodas G. Incidence of tendinopathy in team sports in a multidisciplinary sports club over 8 seasons. J Sports Sci Med. 2019;18(4):780.
9. Silbernagel Grävare K, Crossley KM. A proposed return-to-sport program for patients with midportion Achilles tendinopathy: rationale and implementation. J Orthop Sports Phys Ther. 2015;45(11):876-886.
10. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med. 2007;35(6):897-906.
11. Magnussen RA, Dunn WR, Thomson AB. Nonoperative treatment of midportion Achilles tendinopathy: a systematic review. Clin J Sport Med. 2009;19(1):54-64.
12. Brown R, Orchard J, Kinchington M, Hooper A, Nalder G. Aprotinin in the management of Achilles tendinopathy: a randomised controlled trial. Br J Sports Med. 2006;40(3):275-279.
13. Mafi N, Lorentzon R, Alfredson H. Superior short-term results with eccentric calf muscle training compared to concentric training in a randomized prospective multicenter study on patients with chronic Achilles tendinosis. Knee Surg Sports Traumatol Arthrosc. 2001;9:42-47.

14. Roos EM, Engström M, Lagerquist A, Söderberg B. Clinical improvement after 6 weeks of eccentric exercise in patients with mid-portion Achilles tendinopathy–a randomized trial with 1-year follow-up. Scand J Med Sci Sports. 2004;14(5):286-295.
15. de Vos RJ, van der Vlist AC, Zwerver J, et al. Dutch multidisciplinary guideline on Achilles tendinopathy. Br J Sports Med. 2021;55(20):1125-1134.
16. Leadbetter WB, Buckwalter JA, Gordon SL. Sports-induced inflammation: clinical and basic science concepts. (No Title). Published online 1990.
17. Kettunen JA, Kvist M, Alanen E, Kujala UM. Long-term prognosis for Jumper's knee in male athletes: prospective follow-up study. Am J Sports Med. 2002;30(5):689-692.
18. Swenson DM, Collins CL, Best TM, Flanigan DC, Fields SK, Comstock RD. Epidemiology of knee injuries among US high school athletes, 2005/06–2010/11. Med Sci Sports Exerc. 2013;45(3):462.
19. Lian ØB, Engebretsen L, Bahr R. Prevalence of jumper's knee among elite athletes from different sports: a cross-sectional study. Am J Sports Med. 2005;33(4):561-567.
20. Jildeh TR, Buckley P, Abbas MJ, et al. Impact of patellar tendinopathy on player performance in the National Basketball Association. Orthop J Sports Med. 2021;9(9):23259671211025305.
21. Gemignani M, Busoni F, Tonerini M, Scaglione M. The patellar tendinopathy in athletes: a sonographic grading correlated to prognosis and therapy. Emerg Radiol. 2008;15:399-404.
22. Lempainen L, Johansson K, Banke IJ, et al. Expert opinion: diagnosis and treatment of proximal hamstring tendinopathy. Muscles Ligaments Tendons J. 2015;5(1):23.
23. Lempainen L, Sarimo J, Mattila K, Vaittinen S, Orava S. Proximal hamstring tendinopathy: results of surgical management and histopathologic findings. Am J Sports Med. 2009;37(4):727-734.

Nutrition Strategies for the Injured Tendon

Take Home Points

- Adequate energy intake, fluid intake, and protein consumption are crucial for effective healing and recovery from tendinopathy. Ensuring a balanced diet with sufficient nutrients can mitigate muscle and tendon mass loss, enhance wound healing, and expedite the return to sports activities.
- Proper hydration, monitored through various indicators, is essential to facilitate nutrient delivery for healing.
- Protein intake plays a pivotal role in collagen production, muscle protein synthesis, and wound healing. Athletes recovering from tendinopathy should aim for increased protein consumption, consuming it evenly throughout the day.
- Vitamin C is vital for collagen formation and can be obtained from various food sources to support tendon health. Other supplements, such as gelatin, may also support collagen synthesis and healing.
- There is little consensus in the literature regarding the use of dietary supplements for tendon healing. However, a supplement protocol of 15g of collagen plus 50 mg of vitamin C ingested 60 minutes prior to physical activity or rehabilitation may be beneficial and is of little risk, provided athletes choose products with appropriate third-party certifications.

During treatment and rehabilitation of tendinopathy, attempting to prevent a deficiency of daily energy availability, ensuring adequate fluid and amino acid intake, and satisfying vitamin C needs is critical.[1] By doing so, an athlete can reduce the potential for significant losses of muscle and tendon mass and thus functional declines and improve wound healing, thereby accelerating the return to sport process. Secondarily, nutrients and supplements may confer additional benefits on tendon healing, though scientific consensus is still lacking in this area. Conversely, alcohol intake should be avoided or minimized, as it impairs muscle protein synthesis and wound healing[2] and may inhibit collagen synthesis.[1,3] Given the paucity of evidence-based recommendations for nutritional interventions specific to tendinopathy, the following section includes supporting literature, suggested best-practices, and recommendations from registered dieticians at the United States Olympic and Paralympic Committee.

Energy Balance

During the injury recovery period, it is anecdotally common for athletes to reduce nutritional intake - either intentionally to prevent body weight and composition changes or

unintentionally as a response to decreased appetite and/or medication-related gastrointestinal side effects. However, this can have significant consequences on wound healing and muscle and tendon tissue loss. Energy needs are dynamic and can be challenging to estimate during periods of extended inactivity, especially in cases of severe injuries and/or surgery where the body may be in a hypermetabolic state. While exercise expenditure drops when an athlete is immobilized or observing training restrictions, overall energy expenditure may, in fact, increase temporarily by 15-50% due to the natural inflammatory and healing processes, dependent on the severity of the insult.[4] Additionally, if an athlete is ambulating with crutches or a wheelchair, they may be expending a great deal of energy from their mode of transportation.[1] Of course, overestimating energy intake can lead to body weight and composition changes that make returning to play more difficult, add unnecessary load to the tendon, and can affect the athlete's body image and confidence. A registered dietitian (RD) can estimate individualized energy needs, which is a critical part of the collaborative care process. The following guidelines can be a starting point for RDs in estimating needs during recovery, as discussed by Dana White in a webinar available online:[5]

1. Calculate baseline needs for weight maintenance: 30 calories/kg of body weight.
2. Add additional calories to the baseline based on the severity of injury:
    - 5-10 calories/kg/day for moderate injuries (such as grade 1 strains/sprains)
    - 10-15 calories/kg/day for significant trauma (such as grade 2 tears)
    - 25-30 calories/kg/day for severe trauma (such as grade 3 tears/ruptures or surgery)

Fluid Intake

Tendons are made of approximately 65-75% water and some are also surrounded by a water-rich synovial fluid.[6] Tendons absorb nutrients through bulk fluid flow; fluid flows outward when tendons are loaded and fluid is drawn inward as they relax.[7] Thus, activity encourages the flow of blood and nutrients into tendon tissue. Therefore, adequate fluid intake, especially before and during rehabilitation activity, is an area of opportunity for injured athletes to maximize nutrient delivery for healing. Athletes can monitor hydration status by being mindful of urine color, void frequency, urine volume, thirst, and drinking a variety of fluids consistently throughout the day. Furthermore, an RD may recommend an athlete monitor weight changes pre- and post-training/rehabilitation to identify individualized hydration needs for recovery. Optimal hydration status can be achieved by relatively simple yet effective daily (List 1)[8] and exercise-related (List 2) strategies.

List 1. Simple methods for increasing daily fluid intake.

- Carry a water bottle at all times to increase water consumption throughout the day.

- Aim to drink at least 2 cups of water at all meals.
- Fruit and veggies have high water content.
- Snack on oranges, berries, melons, pineapple.
- Top a rice bowl with eggplant, bell peppers, zucchini, and shredded carrots.
- Make a fruit smoothie for breakfast or a snack.
- Drink 8 oz. of water, milk, 100% fruit juice, herbal tea, or coconut water with breakfast.
- Begin lunch or dinner with veggie soup.
- Drink a glass of milk after training or before bed.
- Brew a cup of herbal tea in the evening.

List 2. Peri-exercise strategies for optimal hydration*

Before Exercise

- Begin exercise well-hydrated
- Drink 16-20 oz. of water or sports beverage at least four hours before exercise
- Drink 8-12 oz. of water 10-15 min. before exercise.

During Exercise

- Drink water or sports beverage every 15-20 min. during exercise
- 3-8 oz. of water (2-3 large gulps) for exercise <60 mins.
- 3-8 oz. of sports beverage for exercise >60 mins.

After Exercise

- Rehydrate
- 16-24 oz. of fluid for every pound lost within 2 hours of exercise.
- Chocolate milk is a great option to help rehydrate and refuel after a workout.
-

*Adapted from [https://www.sportsrd.org/wp-content/uploads/2016/01/Hydration-Screen-Shot-.png].

P*rotein & Amino Acids*

Being mindful of protein intake during the recovery period is a critical piece of injury recovery. Increasing relative protein intake enables essential amino acids to facilitate collagen production and muscle protein synthesis. Insufficient protein intake will impede wound healing.[9] At a minimum, athletes should aim for 1.6 g of protein per kg of body mass.[1,10] If an athlete's habitual intake is already meeting that minimum, they may benefit from a further increase to 2.0 g/kg or more, especially if immobilized.[1,5,10] Relative to the timing of meals, distributing protein intake uniformly across the day is preferred.[10]

Proteins from plant and animal sources differ in their amino acid profiles, and athletes benefit greatly from incorporating a variety in their choices. For example, whey protein (from

cow's milk-based food and protein products) is rich in the amino acid leucine, which has been suggested to exhibit a stimulatory effect on collagen synthesis in response to resistance exercise.[3,11] Red meat, turkey, chicken, and nuts contain lysine, glycine, and proline, which are central to collagen formation when combined with vitamin C. Red meat, turkey, chicken, nuts, fish, and beans/legumes are sources of l-arginine, an amino acid involved in the production of nitric oxide, which may also facilitate healing.[12-14] RDs can help athletes meet their individual protein needs and ensure the consumption of a variety of amino acids through their habitual diet with respect to personal preferences, access, and budget.

Vitamin C

Vitamin C plays a central role in the formation of collagen,[1,7,15,16] the most abundant protein in connective tissue.[7] Vitamin C is most beneficial in food form, and injured athletes would benefit from incorporating rich sources of vitamin C into meals and snacks. All fruits and vegetables contain vitamin C, with bell peppers, orange juice, whole citrus fruits, kiwi, and berries having the greatest vitamin C density among whole-food sources. If athletes have difficulty in meeting their needs through food, a supplement may be utilized, with the understanding that high-dose supplementation beyond sufficiency does not appear to have additional benefits for tendon healing.[1] The Recommended Dietary Allowance (RDA) for vitamin C is 75mg and 65mg for males and females, respectively, for ages 14-18, and 90mg and 75mg for males and females, respectively, for ages 19 and above.[17]

Nitric Oxide & Additional nutrients

Nitric oxide (NO) is an essential factor in the tendon healing process,[15] with both animal and human studies showing that its production is upregulated in tendinopathy.[18] NO assists in the healing process via multiple mechanisms, including assisting in collagen synthesis.[19] Some studies have suggested that dietary supplements and topical patches may be effective at upregulating and stimulating NO synthesis.[18] Incorporating foods that stimulate NO production, namely beets and beet products (juices, powders) and dark, green leafy vegetables like spinach, arugula, and Swiss chard, could form part of a dietary program during tendinopathy treatment and recovery. Several whole-food sources and supplements may also be effective for tendon health and wound healing (Table 1), and athletes are encouraged to consult an RD and discuss whether the inclusion of any foods or supplements would be beneficial for their specific needs. Regardless, athletes should focus on food variety and the inclusion of abundant protein, fruits, and vegetables during recovery from tendinopathy.

Table 1. Food sources of Key Nutrients for Healthy and Injured Tendons[19]

Nutrient	Sources	Important for tendon/ligament health	Important for muscle health	Important for wound healing*

Nitric oxide	Beets, spinach arugula, celery radishes, swiss chard	✅		
Leucine	Chicken, beef, fish, pork, egg, dairy, soy	✅	✅	
HMB	Leucine-rich foods such as whey protein, chicken, beef, fish, pork, egg, dairy, soy		✅	
CoQ10	Beef, chicken, trout, soybean oil, olive oil, peanuts, seasme seeds		✅	
Creatine	Wild game, red meat poultry, fish		✅	
Glutamine	Beef, chicken, fish, beans, dairy, cabbage, beets, legumes	✅		✅
Arginine	Shrimp, white mean turkey, frozen spinach			✅
Vitamin A	Sweet potato, carrot, mange, red pepper, cantaloupe, egg yolk, dairy, green vegetables, fish	✅		✅
Vitamin C	Citrus fruit, pineapple, bell peppers, kiwi, broccoli, berries, baked potato, tomato, leafy greens	✅		✅
Omega 3 fatty acids	Fatty fish (salmon, mackeral, sardines, tina, trout), flaxseed, walnuts, canola oil		✅	✅
Zinc	Oysters, beef, fortified cereals, pork, beans, dark meat chicken, yogurt, cashews, chickpeas		✅	✅

*Utilize these nutrients if healing from a surgical intervention to repair a soft tissue injury.

Dietary Supplements

A wide variety of supplements have been proposed to heal tendons, increase collagen production, reduce pain, or accelerate recovery from injury. There is little consensus in the scientific literature on the most effective products and protocols due in part to the challenge of isolating specific nutrients in human trials[6,15] and the ability to conduct research on elite athletes. Further, Team USA athletes (and other elite athlete groups) are subject to testing for banned substances and, therefore, should only be recommended products that have a reliable third-party testing certification, such as NSF Certified for Sport or Informed Sport.

Collagen + Vitamin C

One of the most widely studied supplements for specific use in tendinopathy treatment is collagen. When co-ingested with vitamin C, collagen has been shown to increase circulating amino acids that are critical to forming the collagen matrix,[3,7,11,15,16,21,22] reduce pain,[11,16,21] and improve joint functionality.[16] Because blood flow to inactive tendons is limited but appears to increase during activity, supplement protocols should consider the timing of the supplementation just as rigorously as the dosage. As such, a supplement protocol of 15g of

collagen and 50 mg of vitamin C ingested 60 minutes prior to physical activity or rehabilitation[7,11,22] may be potentially beneficial for tendon health and recovery. As always, athletes are encouraged to discuss their specific needs with a trusted RD.

Similarly, gelatin, a hydrolyzed form of collagen, is a popular and inexpensive food product that forms a gel-like substance in water. Gelatin can be used to make homemade jellies with the same benefits as more expensive collagen products. Recipes for gelatin are readily available, but an example of this simple and inexpensive supplement, adapted from a recipe suggested by prominent collagen researcher Dr. Keith Baar[22] is found in below.

Ingredients:

160g (1 cup) gelatin (commonly seen in grocery stores as Knox brand)
12 oz (1.5 cups) high vitamin C juice (like Juicy Juice 100% juice products or Ribena)
8 oz (1 cup) water

Directions:

Lightly spray a 9x9 inch baking dish with non-stick spray.
Bring juice to a boil.
Meanwhile, mix gelatin into water.
Add the hot juice to the gelatin and stir until dissolved.
Pour gelatin into the baking dish and place in fridge until set.
Cut into 5 portions. Eat 1 portion 60 minutes before tendon-stimulating activity.

Additional Supplements of Interest

Many other products have been mentioned in the literature specific to tendon healing but are limited in research, including whey protein, leucine, omega 3s, creatine, l-arginine, curcumin, glucosamine, and chondroitin.[1,3,15,18] Products such as whey protein powder may secondarily support tendon recovery by helping athletes meet their protein needs. Athletes can work with their RDs and sports medicine teams to determine if these options would be safe and beneficial for their dietary needs.

References

1. Tipton KD. Nutritional support for exercise-induced injuries. Sports Med. 2015;45(Suppl 1):93–104.
2. Hapa O, Çakıcı H, Gideroğlu K, Özturan K, Kükner A, Buğdaycı G. The effect of ethanol intake on tendon healing: a histological and biomechanical study in a rat model. Arch Orthop Trauma Surg. 2009;129:1721–6.
3. Hijlkema A, Roozenboom C, Mensink M, Zwerver J. The impact of nutrition on tendon health and tendinopathy: a systematic review. J Int Soc Sports Nutr. 2022;19(1):474–504.
4. Smith-Ryan, Abbie E., Katie R. Hirsch, Hannah E. Saylor, Lacey M. Gould, and Malia N. M. Blue. 2020. "Nutritional Considerations and Strategies to Facilitate Injury Recovery and Rehabilitation." Journal of Athletic Training 55 (9): 918–30.
5. Dana White. Sports Nutrition for the Injured Athlete [Internet]. [cited 2024 Mar 25]. Available from: https://www.mysportsd.com/products/101470-Sports-Nutrition-for-the-Injured-Athlete
6. Benjamin M, Ralphs J. Invited Review Tendons and ligaments-an overview. Histol Histopathol. 1997;12:1135–44.
7. Baar K. Training and nutrition to prevent soft tissue injuries and accelerate return to play. Sports Sci Exch. 2015;28(142):1–6.
8. United States Olympic and Paralympic Committee. Hydration [Factsheet]. Hydration [Factsheet]. Available from: https://www.usopc.org/nutrition#:~:text=Fueling%20for%20Competition-,Hydration,-Immune%20System
9. Demling RH. Nutrition, anabolism, and the wound healing process: an overview. Eplasty. 2009;9:e9.
10. Wall BT, Morton JP, Van Loon LJ. Strategies to maintain skeletal muscle mass in the injured athlete: nutritional considerations and exercise mimetics. Eur J Sport Sci. 2015;15(1):53–62.
11. Baar K. Minimizing injury and maximizing return to play: Lessons from engineered ligaments. Sports Med. 2017;47:5–11.
12. Murrell G, Szabo C, Hannafin J, Jang D, Dolan M, Deng XH, et al. Modulation of tendon healing by nitric oxide. Inflamm Res. 1997;46:19–27.
13. Lin J, Wang M, Wei A, Zhu W, Diwan AD, Murrell GA. Temporal expression of nitric oxide synthase isoforms in healing Achilles tendon. J Orthop Res. 2001;19(1):136–42.
14. Lin J, Wang MX, Wei A, Zhu W, Murrell G. The cell specific temporal expression of nitric oxide synthase isoforms during Achilles tendon healing. Inflamm Res. 2001;50:515–22.

15. Fusini F, Bisicchia S, Bottegoni C, Gigante A, Zanchini F, Busilacchi A. Nutraceutical supplement in the management of tendinopathies: a systematic review. Muscles Ligaments Tendons J. 2016;6(1):48.
16. Khatri M, Naughton RJ, Clifford T, Harper LD, Corr L. The effects of collagen peptide supplementation on body composition, collagen synthesis, and recovery from joint injury and exercise: a systematic review. Amino Acids. 2021;53(10):1493–506.
17. National Institute of Health - Office of Dietary Supplements. Vitamin C - Fact Sheet for Health Professionals. [cited 2024 Mar 25]. Office of Dietary Supplements - Vitamin C. Available from: https://ods.od.nih.gov/factsheets/VitaminC-HealthProfessional/
18. Murrell GA. Using nitric oxide to treat tendinopathy. Br J Sports Med. 2007;41(4):227–31.
19. Witte MB, Thornton FJ, Efron DT, Barbul A. Enhancement of fibroblast collagen synthesis by nitric oxide. Nitric Oxide. 2000;4(6):572–82.
20. United States Olympic and Paralympic Committee. Nutrients for Soft Tissue Recovery [Factsheet]. Nutrients for Soft Tissue Recovery [Factsheet]. Available from: https://assets.contentstack.io/v3/assets/blt9e58afd92a18a0fc/blt1982a2b1e73ee5c2/6470c42aec22332ae051b3af/Nutrients_For_Soft_Tissue_Injury_Recovery_Factsheet.pdf
21. Maughan RJ, Burke LM, Dvorak J, Larson-Meyer DE, Peeling P, Phillips SM, et al. IOC consensus statement: dietary supplements and the high-performance athlete. Int J Sport Nutr Exerc Metab. 2018;28(2):104–25.
22. Shaw G, Lee-Barthel A, Ross ML, Wang B, Baar K. Vitamin C–enriched gelatin supplementation before intermittent activity augments collagen synthesis. Am J Clin Nutr. 2017;105(1):136–43.

General Tendinopathy Management Guidelines

Specific tendinopathy exercise prescription recommendations often change from tendon to tendon, but in general, standard treatment "themes" can be seen regardless of the tendon at interest. Certainly the stage of healing, reactivity of the tendon to load, and tendon location will largely shape both short-term, and long-term, exercise programming. While this section will discuss rehab principles based on contraction types, the authors would like to stress these do not need to be prescribed in any particular order. For example, although isometrics are often first prescribed in the presence of pain, isometrics may not necessarily elicit an adaptation in the case of every tendinopathy. As such, the clinician should be conscious of consequences with both underloading and overloading the tendon of interest. Ultimately the loading magnitude seems to be the key factor for tendon adaptation first and foremost rather than contraction type.[1,2]

An adaptation from Millet at al, 2021 is present in "An Overview of the Injured Tendon" section on Page 49. This adaptation includes a Venn-Diagram where contraction-types are coupled with one another only to be followed by energy storage loading and release programming.[3] This figure is particularly useful to the treating clinician as staging of the tendinopathy isn't required. Rather, the initial evaluation should include a comprehensive differential diagnosis and consideration of the nine core health-related domains of tendinopathy recently decided by the ICON 2019 consensus guidelines.[4] These domains were primarily selected to guide future research, but also act in guiding the treating clinician in their global approach and considerations to tendinopathy management. These can be found summarized under **Table 1.** Following evaluation, the patient should be educated on typical tendinopathy rehab guidelines and basic tendinopathy injury mechanisms. An individualized exercise program is then developed with frequent re-evaluations. Passive interventions may be incorporated as necessary, especially in the tendinopathy recalcitrant to typical rehab strategies. Should difficulties ensue past 12 months, surgical options may be considered.[5]

Table 1. The nine core health-related domains of tendinopathy as decided by 28 healthcare providers (92% of whom with >10 years' experience treating tendinopathy) and 32 patients.[4]

Patient rating of condition	A single assessment numerical evaluation ("rate your tendon status where 100% is no problems and 0% worst

	case scenario"), global rating of change (how are you now compared with prior treatment?)[6–8]
Participation in life activities	A patient rating of the level of participation / engagement across areas of their life (i.e. ratings of level of sport, time to return to sport)[9]
Pain on activity / loading	Patient reported intensity of pain on performing a task/activity that loads the tendon (visual analog scale, numeric pain rating scale)[10–12]
Function	Patient rated level of function (Patient Specific Function Scale on a visual analog scale or numeric pain rating scale)[7]
Psychological Factors	Pain self-efficacy, pain catastrophization, kinesiophobia, anxiety or depression scales[7]
Physical Function Capacity	Quantitative measures of physical tasks performed in clinic (i.e. number of hops, timed stair walk, number of single limb squats, dynamometry, etc.)[7,13,14]
Disability	Composite scores of patient-rated pain and disability due to pain usually in relation to tendon specific activities / tasks[7,8,11,12]
Quality of Life	The general well being of the individual questionnaires[7,8,15]
Pain over a Specified Time	Participant reported pain intensity over a period of time (morning, night, 24 hours, a week; visual analog scale, numeric rating scale)[15–17]

A tendinopathy pain-monitoring model originally described by Thomeé,[18] and later modified by Silbernagel et al,[19,20] has been popularized in recent years in helping both clinicians and patients gauge how appropriate a level of tendon pain is, while rehabilitating. On a visual analog scale (VAS) where 0 is no pain and 10 is the worst pain imaginable, pain is allowed to reach a 5 while exercise training.[19,20] Pain after rehabilitation should subside by the following morning.[19,20] It is important to note that this pain-monitoring model was originally published in relation to Achilles tendinopathy, and although helpful, should be used with caution in the application of other tendinopathies. The authors of this publication believe it to still be appropriate given guidelines in the management of most tendinopathies, although it may need to be modified based on the patient's tolerance to load.

Isometrics

An isometric exercise is a form of muscle contraction where tension is built, but skeletal muscle fibers don't change length. Isometrics, which have become popular in recent years in the management of tendinopathy,[5] are celebrated for their utility in post-operative neuromuscular re-education and may provide generalized pain inhibition.[2,21] The percentage of motor unit recruitment is greater for isometric contractions compared to eccentric or concentric exercises so they provide a means to strengthen muscle and enhance neuromuscular activation when joint motion and external loading strategies may be limited due to injury or pain.[2] Further, isometric

loading may be preferable for short-term tendon pain management over other forms of exercise loading, in particular for the in-season athlete.[22] Dosage should be based on symptom severity and irritability, but researchers have advocated for 5 sets of 45-second holds of moderate-resistance isometric exercise performed at 70% maximum voluntary isometric contraction.[21] These recommendations were initially proposed for patellar tendinopathy[21] and clinicians should extrapolate with caution when applying these exercise parameters to other tendons.

Isometric exercise in the management of tendinopathy has been challenged in recent years with conflicting results reported in terms of immediate and short-term pain relief.[5] A 2020 systematic review and meta-analysis determined that isometric exercise does not appear to be superior to isotonic exercise in the management of chronic tendinopathies at other joints.[5] Another 2020 publication found isometric exercises to not have additional benefit when combined with eccentrics over a 3-month intervention period in the management of Achilles tendinopathy.[23] Despite the immediate or short-term analgesic effects of isometric exercise being variable, it may certainly still be used as part of a progressive loading program in some individuals.[5] It is also important to note that often times isometrics are not prescribed in isolation, and are typically threaded in a comprehensive tendinopathy rehab program amongst other contraction types.

Isotonics and Eccentrics

For athletes who started initially with isometric programming, the tendon of focus should soon improve to tolerate the demands of both concentric and eccentric contractions**.** Isotonic exercise involves movement with constant external resistance. Many forms of external resistance can be applied including gravity, dumbbells, barbells and so forth.[24] Many athletes may be able to forgo isometrics and focus the start of their rehabilitation primarily on isotonics. The intent of isotonic programming begins to focus on restoring involved soft tissue strength, bulk, and capacity in a functional range of motion.[5] Heavy, slow, fatiguing resistance training should be introduced as soon as possible.[25] As to what constitutes a “heavy” load is highly variable from athlete to athlete, and will likely need to be individualized.[25]

The eccentric portion of movement occurs when the force applied to the muscle exceeds the force produced by the muscle itself, resulting in a lengthening muscle contraction against resistance.[7] Eccentric exercise is an essential part of tendon loading protocols, however, it is a common rehabilitative pitfall to prescribe them in isolation.[25] Many modern rehab tendinopathy programs often thread eccentrics amongst other contraction types listed in this section. Eccentrics have historically been celebrated for their theorized benefits specifically in tendinopathy management.[26] They have been suggested to influence positive changes at the cellular level through increased fibroblast activity and collagen synthesis in injured tissue.[8,9] In

some cases, these structural changes have been correlated with decreased pain during activity secondary to a decreased release of pain-inducing factors.[9] These proposed benefits aside, other publications in past decade or so have begun to encourage a greater focus on the magnitude of loading instead.[1,2]

Various recommendations exist across the literature regarding eccentrics depending on the tendon of interest.[25] Their application is occasionally contraindicated depending on the portion of the tendon affected. This is especially evident at the calcaneal insertion of the Achilles tendon, where compressive forces are greatest into end-range dorsiflexion.[27,28] Although well-intended, eccentrics for the Achilles are often prescribed here with the forefoot elevated, encouraging irritable compressive forces at end-ranges of dorsiflexion. A similar situation occurs at the proximal hamstring tendon and ischial tuberosity with hamstring exercise programming in deep hip flexion.[25] Further specific considerations will be explored in length at the tendon specific sections toward the end of this manual.

With the aforementioned cautions in consideration and when determined appropriate, recommended eccentric programming typically begins with bodyweight load for 3 sets of 15 repetitions, performed twice a day each day, with progressive increases in load as pain diminishes.[10] Eccentrics are often threaded among other heavy, slow resistance training, which has similar benefits.[10] It is important to recognize that compared to other contraction types, eccentrics allow for the highest magnitudes of mechanical loading.[29] Given this, the contraction type itself may provide less of the actual intended benefits, while the external load allowed by it provides the proposed benefits in the treatment of tendinopathy.

As discussed in the Basic Science and Conceptual Load Management Theories section, acute exercise in humans is followed by an increase in both collagen synthesis and degradation of collagen[30]. A net synthesis of collagen eventually occurs after 36 to 72 hours after exercise. Repeated training, or sessions of rehabilitation, without adequate rest in-between (24 hours) may result in a net degradation of the matrix and may lead to further injury to the tendon.[30–33] This consideration should be at the forefront of the clinician's mind considering eccentric training often leads to more soreness than other exercise modes.[34]

Energy Storage and Release (Plyometrics)

The final stage of tendinopathy programming will primarily focus on incorporating energy storage and release principles.[35] Of all the aforementioned contraction types, energy storage and release drills and activities tend to be the most provacative for a tendon.[25] This phase should only be started once previous stages have been fully established as pain-free. This phase should involve lateral, rotational, and cutting movements to graduate loads in multiple

planes of movement. During this phase, heavy slow resistance training, isometrics,[25] and sport-specific / return to play drills are often prescribed concurrently.

References

1. Bohm S, Mersmann F, Arampatzis A. Human tendon adaptation in response to mechanical loading: a systematic review and meta-analysis of exercise intervention studies on healthy adults. Sports Med Open. 2015;1(1):7.
2. Lim HY, Wong SH. Effects of isometric, eccentric, or heavy slow resistance exercises on pain and function in individuals with patellar tendinopathy: A systematic review. Physiother Res Int. 2018;23(4):e1721.
3. Millar NL, Silbernagel KG, Thorborg K, et al. Tendinopathy. Nat Rev Dis Primers. 2021;7(1):1.
4. Vicenzino B, de Vos RJ, Alfredson H, et al. ICON 2019-International Scientific Tendinopathy Symposium Consensus: There are nine core health-related domains for tendinopathy (CORE DOMAINS): Delphi study of healthcare professionals and patients. Br J Sports Med. 2020;54(8):444-451.
5. Clifford C, Challoumas D, Paul L, Syme G, Millar NL. Effectiveness of isometric exercise in the management of tendinopathy: a systematic review and meta-analysis of randomised trials. BMJ Open Sport Exerc Med. 2020;6(1):e000760.
6. Brinks A, van Rijn RM, Willemsen SP, et al. Corticosteroid injections for greater trochanteric pain syndrome: a randomized controlled trial in primary care. Ann Fam Med. 2011;9(3):226-234.
7. Mellor R, Bennell K, Grimaldi A, et al. Education plus exercise versus corticosteroid injection use versus a wait and see approach on global outcome and pain from gluteal tendinopathy: prospective, single blinded, randomised clinical trial. BMJ. Published online May 2, 2018:k1662.
8. Munteanu SE, Scott LA, Bonanno DR, et al. Effectiveness of customised foot orthoses for Achilles tendinopathy: a randomised controlled trial. Br J Sports Med. 2015;49(15):989-994.
9. Horstmann T, Jud HM, Fröhlich V, Mündermann A, Grau S. Whole-body vibration versus eccentric training or a wait-and-see approach for chronic Achilles tendinopathy: a randomized clinical trial. J Orthop Sports Phys Ther. 2013;43(11):794-803.
10. Rio E, van Ark M, Docking S, et al. Isometric contractions are more analgesic than isotonic contractions for patellar tendon pain. Clin J Sport Med. 2017;27(3):253-259.
11. Thijs KM, Zwerver J, Backx FJG, et al. Effectiveness of shockwave treatment combined with eccentric training for patellar tendinopathy: A double-blinded randomized study. Clin J Sport Med. 2017;27(2):89-96.
12. Beyer R, Kongsgaard M, Hougs Kjær B, Øhlenschlæger T, Kjær M, Magnusson SP. Heavy Slow Resistance Versus Eccentric Training as Treatment for Achilles Tendinopathy: A Randomized Controlled Trial. Am J Sports Med. 2015;43(7):1704-1711.

13. Knobloch K. Re: Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med. 2007;35(7):1208; author reply 1208-9.
14. Zwerver J, Hartgens F, Verhagen E, van der Worp H, van den Akker-Scheek I, Diercks RL. No effect of extracorporeal shockwave therapy on patellar tendinopathy in jumping athletes during the competitive season: a randomized clinical trial. Am J Sports Med. 2011;39(6):1191-1199.
15. Coombes BK, Bisset L, Brooks P, Khan A, Vicenzino B. Effect of corticosteroid injection, physiotherapy, or both on clinical outcomes in patients with unilateral lateral epicondylalgia: a randomized controlled trial. JAMA. 2013;309(5):461-469.
16. Stasinopoulos D, Stasinopoulos I. Comparison of effects of eccentric training, eccentric-concentric training, and eccentric-concentric training combined with isometric contraction in the treatment of lateral elbow tendinopathy. J Hand Ther. 2017;30(1):13-19.
17. Yelland MJ, Sweeting KR, Lyftogt JA, Ng SK, Scuffham PA, Evans KA. Prolotherapy injections and eccentric loading exercises for painful Achilles tendinosis: a randomised trial. Br J Sports Med. 2011;45(5):421-428.
18. Thomeé R. A comprehensive treatment approach for patellofemoral pain syndrome in young women. Phys Ther. 1997;77(12):1690-1703.
19. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med. 2007;35(6):897-906.
20. Silbernagel KG, Crossley KM. A Proposed Return-to-Sport Program for Patients With Midportion Achilles Tendinopathy: Rationale and Implementation. J Orthop Sports Phys Ther. 2015;45(11):876-886.
21. Rio E, Kidgell D, Purdam C, et al. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. Br J Sports Med. 2015;49(19):1277-1283.
22. Rio E, Purdam C, Girdwood M, Cook J. Isometric exercise to reduce pain in patellar tendinopathy in-season; Is it effective “on the road?” Clin J Sport Med. Published online November 16, 2017. doi:10.1097/JSM.0000000000000549
23. Gatz M, Betsch M, Dirrichs T, et al. Eccentric and isometric exercises in Achilles tendinopathy evaluated by the VISA-A score and shear wave elastography. Sports Health. 2020;12(4):373-381.
24. Baechle, TR, Earle, RW, and Wathen, D. Essentials of Strength Training and Conditioning (3rd Edition). Champaign, IL: Human. Human Kinetics; 2008.
25. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf

26. Irby A, Gutierrez J, Chamberlin C, Thomas SJ, Rosen AB. Clinical management of tendinopathy: A systematic review of systematic reviews evaluating the effectiveness of tendinopathy treatments. Scand J Med Sci Sports. 2020;30(10):1810-1826.
27. Cook JL, Purdam C. Is compressive load a factor in the development of tendinopathy? Br J Sports Med. 2012;46(3):163-168.
28. de Vos RJ, van der Vlist AC, Zwerver J, et al. Dutch multidisciplinary guideline on Achilles tendinopathy. Br J Sports Med. 2021;55(20):1125-1134.
29. Hortobágyi T, Katch FI. Eccentric and concentric torque-velocity relationships during arm flexion and extension. Influence of strength level. Eur J Appl Physiol Occup Physiol. 1990;60(5):395-401.
30. Magnusson SP, Langberg H, Kjaer M. The pathogenesis of tendinopathy: balancing the response to loading. Nat Rev Rheumatol. 2010;6(5):262-268.
31. Miller BF, Olesen JL, Hansen M, et al. Coordinated collagen and muscle protein synthesis in human patella tendon and quadriceps muscle after exercise. J Physiol. 2005;567(Pt 3):1021-1033.
32. Langberg H, Skovgaard D, Karamouzis M, Bülow J, Kjaer M. Metabolism and inflammatory mediators in the peritendinous space measured by microdialysis during intermittent isometric exercise in humans. J Physiol. 1999;515 (Pt 3)(Pt 3):919-927.
33. Langberg H, Skovgaard D, Petersen LJ, Bülow J, Kjær M. Type I collagen synthesis and degradation in peritendinous tissue after exercise determined by microdialysis in humans. J Physiol. 1999;521(1):299-306.
34. Serinken MA, Gençoğlu C, Kayatekin BM. The effect of eccentric exercise-induced delayed-onset muscle soreness on positioning sense and shooting percentage in wheelchair basketball players. Balkan Med J. 2013;30(4):382-386.
35. Cook JL, Docking SI. “Rehabilitation will increase the ‘capacity’ of your …insert musculoskeletal tissue here….” Defining ‘tissue capacity’: a core concept for clinicians. Br J Sports Med. 2015;49(23):1484-1485.

Specific Tendinopathy Management

Patellar Tendinopathy

Symptom Patterns[1]

- Pain localized to the inferior pole of the patella (**Figure 1**)
- Pain that increases with knee extensor muscle loading, specifically during dynamic, plyometric-type movements that require rapid storage and release of energy through muscle contraction
- Pain during loading activities rather than rest or static seated positions

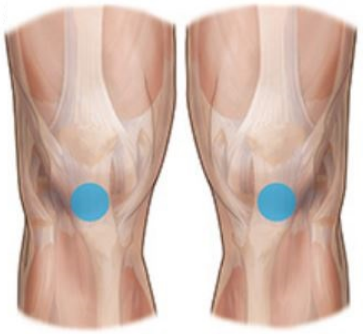

Figure 1: Typical pain location associated with patellar tendinopathy. Illustration with permission from Barça Innovation Hub 2021 Tendon Guide.[2]

Causative Factors[1,3–7]

- Decreased quadriceps and hamstring flexibility[1,4,5]
- Abnormal patellar tracking patterns[8]
- Altered landing biomechanics, thereby resulting in increased ground reaction forces[3,6]
- Greater activity volumes[7]
- Higher body weights[7]
- Greater counter-jump movement height[7]

Movement Pattern Considerations

Hip versus knee dominant movement patterning, particularly during squat exercise variations, can influence loading across the knee joint.[9] An acutely, reactive patellar tendinopathy would likely be better served with hip dominant squats, which act to offload the patellar tendon. As the tendon progresses through rehabilitation and becomes more tolerant of load, knee dominant squats become a suitable exercise prescription again.

Tendon Region-Specific Exercise Recommendations[1,10–12]

A recent 2023 publication by Silva et al investigated patellar tendon loading profiles of common rehabilitation exercises.[13] Traditionally, the single-leg decline squat is the most commonly performed exercise for patellar tendinopathy.[14] However, appreciating other

exercises that should precede the single-leg decline squat in terms of loading profiles (peak, impulse, rate) is important, as some patients may not be immediately able to tolerate the single-leg squat. Silva et al. examined 35 common rehabilitation exercises and created loading indices for each exercise to organize into low, moderate and high categories. This is summarized below in **Table 1**. The authors of this manuscript encourage the reader to reference Silva's publication should they wish to see a complete list of the 35 exercises with affiliated descriptive biomechanical data.[13]

Table 1. Patellar tendon loading profiles separated by tiers[13]. Phase to phase progressions should be based upon the athlete's ability to demonstrate a positive response to performing the exercises outlined which considers pain levels, quality of movement and ability to gradually increase loading. Note the context may vary from athlete to athlete based on health-history related variables.

Tier	Loading Profiles
Tier 1 (Mild)	Walking, low step-up, squat to 60^0, low step down, high step up
Tier 2 (Moderate)	Bulgarian squat, sumo squat, single leg squat to 60^0, squat (full depth), high step down, lunge, drop landing drill, spanish squat, single leg squat (full), counter jump movement drill, running
Tier 3 (High)	Single limb maximal forward hop, single limb repeated forward hops, single limb countermovement hop, run and cut, single limb decline squat

As previously mentioned, progressive loading protocols, regardless of contraction type, appear to provide similar improvements in pain and function with patellar tendinopathy.[12,15–18] As such, isometrics, concentrics, and eccentrics can be programmed with one another.[19] Late stage rehab should include energy storage loading and energy storage release.[19,20] Should the clinician wish to seek additional pain-relief for the patient with patellar tendinopathy, isometric knee extensions have been shown to potentially induce an analgesic effect.[11,21] These may be prescribed at the start of a rehabilitation program in hopes the analgesic effects may encourage subsequent patellar tendon loading to be more comfortable. The isometric performed is typically knee extension which can be performed into a wall or a locked knee extension machine.[11,21] Typical parameters include 5 repetitions of :45 seconds each at 70% max effort.[11,21]

References

1. Malliaras P, Cook J, Purdam C, Rio E. Patellar Tendinopathy: Clinical Diagnosis, Load Management, and Advice for Challenging Case Presentations. J Orthop Sports Phys Ther. 2015;45(11):887-898.
2. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
3. Mann KJ, Edwards S, Drinkwater EJ, Bird SP. A lower limb assessment tool for athletes at risk of developing patellar tendinopathy. Med Sci Sports Exerc. 2013;45(3):527-533.
4. Cook JL, Kiss ZS, Khan KM, Purdam CR, Webster KE. Anthropometry, physical performance, and ultrasound patellar tendon abnormality in elite junior basketball players: a cross-sectional study. Br J Sports Med. 2004;38(2):206-209.
5. Witvrouw E, Bellemans J, Lysens R, Danneels L, Cambier D. Intrinsic risk factors for the development of patellar tendinitis in an athletic population: a two-year prospective study. Am J Sports Med. 2001;29(2):190-195.
6. Richards DP, Ajemian SV, Wiley JP, Zernicke RF. Knee joint dynamics predict patellar tendinitis in elite volleyball players. Am J Sports Med. 1996;24(5):676-683.
7. Sprague AL, Smith AH, Knox P, Pohlig RT, Grävare Silbernagel K. Modifiable risk factors for patellar tendinopathy in athletes: a systematic review and meta-analysis. Br J Sports Med. 2018;52(24):1575-1585.
8. Reinking MF. Current concepts in the treatment of patellar tendinopathy. Int J Sports Phys Ther. 2016;11(6):854-866.
9. Fry AC, Smith JC, Schilling BK. Effect of knee position on hip and knee torques during the barbell squat. J Strength Cond Res. 2003;17(4):629-633.
10. Rio E, Purdam C, Girdwood M, Cook J. Isometric exercise to reduce pain in patellar tendinopathy in-season: Is it effective “on the road”? Clin J Sport Med. 2019;29(3):188-192.
11. Rio E, Kidgell D, Purdam C, et al. Isometric exercise induces analgesia and reduces inhibition in patellar tendinopathy. Br J Sports Med. 2015;49(19):1277-1283.
12. Kongsgaard M, Kovanen V, Aagaard P, et al. Corticosteroid injections, eccentric decline squat training and heavy slow resistance training in patellar tendinopathy. Scand J Med Sci Sports. 2009;19(6):790-802.
13. Scattone Silva R, Song KE, Hullfish TJ, Sprague A, Silbernagel KG, Baxter JR. Patellar tendon load progression during rehabilitation exercises: Implications for the treatment of patellar tendon injuries. Med Sci Sports Exerc. 2024;56(3):545-552.

14. Mendonça LDM, Bittencourt NFN, Alves LEM, Resende RA, Serrão FV. Interventions used for Rehabilitation and Prevention of Patellar Tendinopathy in athletes: a survey of Brazilian Sports Physical Therapists. Braz J Phys Ther. 2020;24(1):46-53.
15. Breda SJ, Oei EHG, Zwerver J, et al. Effectiveness of progressive tendon-loading exercise therapy in patients with patellar tendinopathy: a randomised clinical trial. Br J Sports Med. 2021;55(9):501-509.
16. Malliaras P, Barton CJ, Reeves ND, Langberg H. Achilles and patellar tendinopathy loading programmes : a systematic review comparing clinical outcomes and identifying potential mechanisms for effectiveness. Sports Med. 2013;43(4):267-286.
17. Lim HY, Wong SH. Effects of isometric, eccentric, or heavy slow resistance exercises on pain and function in individuals with patellar tendinopathy: A systematic review. Physiother Res Int. 2018;23(4):e1721.
18. Mendonça LDM, Leite HR, Zwerver J, Henschke N, Branco G, Oliveira VC. How strong is the evidence that conservative treatment reduces pain and improves function in individuals with patellar tendinopathy? A systematic review of randomised controlled trials including GRADE recommendations. Br J Sports Med. 2020;54(2):87-93.
19. Millar NL, Silbernagel KG, Thorborg K, et al. Tendinopathy. Nat Rev Dis Primers. 2021;7(1):1.
20. Cook JL, Docking SI. "Rehabilitation will increase the 'capacity' of your …insert musculoskeletal tissue here…." Defining 'tissue capacity': a core concept for clinicians. Br J Sports Med. 2015;49(23):1484-1485.
21. Rio E, van Ark M, Docking S, et al. Isometric contractions are more analgesic than isotonic contractions for patellar tendon pain. Clin J Sport Med. 2017;27(3):253-259.

Achilles Tendinopathy

Symptom Patterns

Achilles tendinopathy can be classified into either **insertional** or **midportion** tendinopathies. While symptoms are somewhat similar, the location of tendinopathy determines the category.

Insertional: Localized within the first 2 cm of the attachment of the Achilles tendon on the calcaneus.[1] Pain with activity and sensations of morning stiffness. Tenderness to palpation and possible thickening in the area of pain.[2]

Midportion: Localized >2 cm above the attachment of the Achilles onto the calcaneus.[1] Pain with activity and sensations of morning stiffness. Tenderness to palpation and possible thickening in the area of pain.[2] The majority of Achilles tendinopathies are located in the midportion of the tendon (55% to 65%).[3,4]

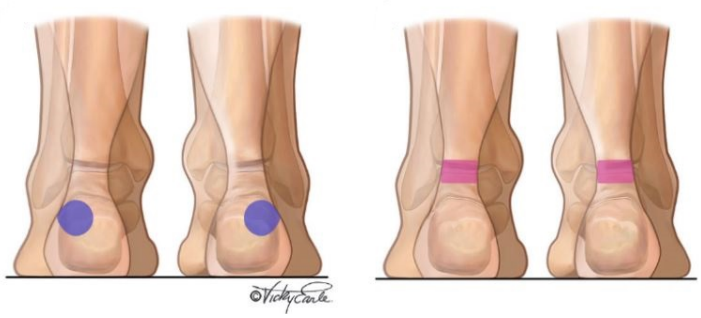

Figure. 1: Typical pain location associated with insertional Achilles tendinopathy (left). A typical pain presentation for midportion Achilles tendinopathy (right). Illustration with permission from Barça Innovation Hub 2021 Tendon Guide.[5]

Causative Factors

- Repetitive overloading of the Achilles tendon and training errors (rapid increases in training intensity or duration)[3,4]
- Chronic diseases that affect tendon quality (i.e. diabetes, rheumatoid arthritis, hypercholesterolemia)[1]
- Use of fluoroquinolones, statins, or moderate alcohol use[6]
- Possibly decreased ankle dorsiflexion[6–8] (although there is conflicting literature[7,9])
- Decreased isokinetic plantar flexion weakness[6]
- Abnormal gait pattern with decreased forward propulsion or increased lateral foot contact during forefoot flat phase[6]

Movement Pattern Considerations

Although similar in clinical presentation, there are clear distinctions in the management of these two conditions.[2] For example, forefoot elevated eccentrics are more likely to aggravate an insertional tendinopathy due to increased compressive loading into increased end-ranges of dorsiflexion. Therefore, exercises are initially advised on a flat surface for those with insertional tendinopathy.[1,10] Midsubstance tendinopathies may better tolerate forefoot elevated programming. If needed, the degree of dorsiflexion can be limited by placing a lift (.5 to 1 in) under the heel.[10]

Tendon Region-Specific Exercise Recommendations

A pain-monitoring model,[11–13] first proposed by Silbernagel et al. in 2007, specifically for Achillies Tendinopathy should be used. (See **Figure 2.**) During activity/rehabilitation, immediately thereafter, or the following morning, the pain should not exceed a "5" on a Numeric Pain Rating Scale of 0-10 (Figure 2). Pain and stiffness should not increase from week to week.

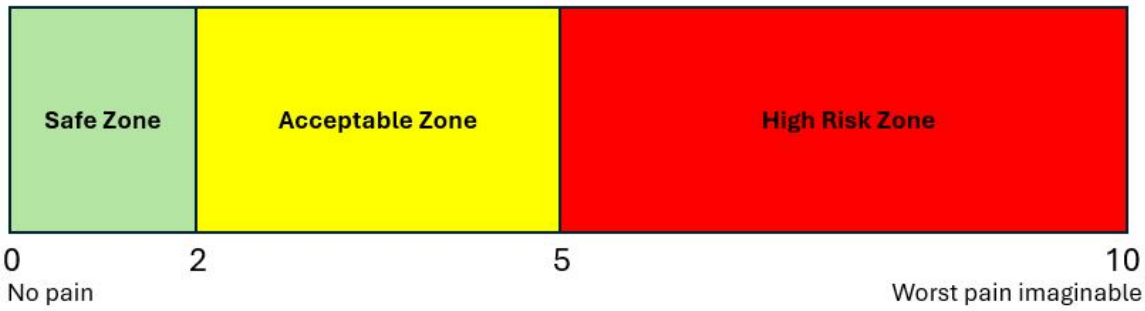

Figure 2. Pain Monitoring Model adapted from Silbernagel et al.[11–13]

As discussed previously, any type of muscle contraction can be used to mechanically load the Achilles tendon. The focus should remain on progressive programming based on the individual patient's response.[10] Exercise rehabilitation continues to remain the treatment with the highest level of evidence in the management of Achilles tendinopathy.[11,14–16] Throughout the course of treatment, various types of heel raises are used for Achilles tendon loading. The patient's degree of symptoms and irritability should be the primary guide for advancing through the rehabilitation protocol.[10] The following summarized protocol was proposed by Silbernagel et al. in a recent 2020 publication.[10] The authors encourage the reader to reference this article for the complete protocol with additional detail.

Phase #1, weeks 1 to 2 (or longer as needed) *May be performed once a day*

Loading Intensity: Progress loading up to 100% body weight with slow, controlled motion
Exercise Examples: Two-legged heel raises on floor (3x10 to 15 repetitions), one-legged heel raises on floor (3x10 repetitions), eccentric heel raises standing on floor (3x10 repetitions), sitting heel raises (3x10 repetitions)

Phase #2, weeks 2 to 5 (or longer as needed) *May be performed once a day*
Loading Intensity: Load on Achilles is progressed by increasing speed of movement and the addition of adding external resistance
Exercise Examples: Two-legged heel raises on edge of step (3x15 repetitions), one-legged heel raises on edge of step (3x15 repetitions), eccentric heel raises standing on edge of step (3x15 repetitions), sitting heel raises (3x15 repetitions), quick-rebounding heel raises (3x20 repetitions)

Phase #3, weeks 3 to 12 (or longer as needed) *Perform once a day and 2-3x/week with heavier load*
Loading Intensity: Continue to progress external resistance and initiate plyometric exercises as appropriate.
Exercise Examples: One-legged heel raises on edge of step with weight (3x15 repetitions), eccentric heel raises standing on edge of step with weight (3x15 repetitions), sitting heel raises with added weight (3x15 repetitions), quick-rebounding heel raises (3x20 repetitions), sport-specific plyometric training

Phase #4, 3 to 6 months (or longer as needed) *Perform exercises 2-3x/week*
Loading Intensity: Progress from previous phase to include sport-specific loading speed and movement patterns on high-intensity days
Exercise Examples: One-legged heel raises on edge of step with added weight (3x15 repetitions), eccentric heel raises standing on edge of step with added weight (3x15 repetitions), quick-rebounding heel raises (3x20 repetitions)

Phase to phase progressions should be based upon the athlete's ability to demonstrate a positive response to performing the exercises outlined which considers pain levels, quality of

movement and ability to gradually increase loading. Note the context may vary from athlete to athlete based on health-history related variables.

References

1. de Vos RJ, van der Vlist AC, Zwerver J, et al. Dutch multidisciplinary guideline on Achilles tendinopathy. Br J Sports Med. 2021;55(20):1125-1134.
2. Silbernagel KG, Crossley KM. A Proposed Return-to-Sport Program for Patients With Midportion Achilles Tendinopathy: Rationale and Implementation. J Orthop Sports Phys Ther. 2015;45(11):876-886.
3. Järvinen TAH, Kannus P, Maffulli N, Khan KM. Achilles tendon disorders: etiology and epidemiology. Foot Ankle Clin. 2005;10(2):255-266.
4. Kvist M. Achilles tendon injuries in athletes. Sports Med. 1994;18(3):173-201.
5. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
6. van der Vlist AC, Breda SJ, Oei EHG, Verhaar JAN, de Vos RJ. Clinical risk factors for Achilles tendinopathy: a systematic review. Br J Sports Med. 2019;53(21):1352-1361.
7. Kaufman KR, Brodine SK, Shaffer RA, Johnson CW, Cullison TR. The effect of foot structure and range of motion on musculoskeletal overuse injuries. Am J Sports Med. 1999;27(5):585-593.
8. Rabin A, Kozol Z, Finestone AS. Limited ankle dorsiflexion increases the risk for midportion Achilles tendinopathy in infantry recruits: a prospective cohort study. J Foot Ankle Res. 2014;7.
9. Mahieu NN, Witvrouw E, Stevens V, Van Tiggelen D, Roget P. Intrinsic risk factors for the development of achilles tendon overuse injury: a prospective study. Am J Sports Med. 2006;34(2):226-235.
10. Silbernagel KG, Hanlon S, Sprague A. Current clinical concepts: Conservative management of Achilles tendinopathy. J Athl Train. 2020;55(5):438-447.
11. Silbernagel KG, Thomeé R, Eriksson BI, Karlsson J. Continued sports activity, using a pain-monitoring model, during rehabilitation in patients with Achilles tendinopathy: a randomized controlled study. Am J Sports Med. 2007;35(6):897-906.
12. Silbernagel KG, Thomeé R, Thomeé P, Karlsson J. Eccentric overload training for patients with chronic Achilles tendon pain--a randomised controlled study with reliability testing of the evaluation methods. Scand J Med Sci Sports. 2001;11(4):197-206.
13. Thomeé R. A comprehensive treatment approach for patellofemoral pain syndrome in young women. Phys Ther. 1997;77(12):1690-1703.
14. Martin RL, Chimenti R, Cuddeford T, et al. Achilles pain, stiffness, and muscle power deficits: Midportion Achilles tendinopathy revision 2018. J Orthop Sports Phys Ther. 2018;48(5):A1-A38.

15. Silbernagel KG, Gustavsson A, Thomeé R, Karlsson J. Evaluation of lower leg function in patients with Achilles tendinopathy. Knee Surg Sports Traumatol Arthrosc. 2006;14(11):1207-1217.
16. Achilles Pain, Stiffness, and Muscle Power Deficits: Midportion Achilles Tendinopathy Revision 2018: Using the evidence to guide physical therapist practice. J Orthop Sports Phys Ther. 2018;48(5):425-426.

Adductor Tendinopathy

Symptom Pattern[1,2]

- Pain that typically presents in the groin or lower abdominal region with occasional referral to the medial thigh
- Pain provocation with daily activities such as climbing stairs, transfers into/out of a bed or car
- Pain with resisted adduction and/or passive abduction
- Pain with sneezing or coughing

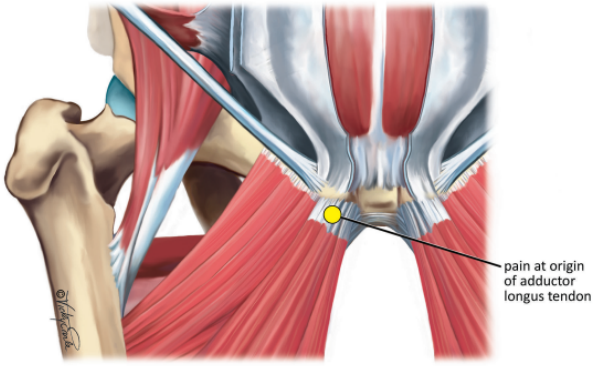

Figure. 1: Typical pain location with Adductor tendinopathy. Illustration with permission from Barça Innovation Hub 2021 Tendon Guide.[3]

Note: Early diagnoses and recognition of adductor tendinopathy is important to appropriately manage this condition given the proximity of several complex anatomical structures in and around the groin region and the broad differential diagnoses for groin pain (e.g., osteitis pubis, sports hernia, iliopsoas bursitis, pelvic stress/avulsion fractures, lumbar spine pathology, and hip impingement).[4]

Causative Factors[1,5]

- Functional overuse caused by torsion and/or traction of the adductor tendon insertion, particularly in sports involving sudden changes of direction, continuous acceleration/deceleration, sliding tackles, and kicking
- Strength imbalance between the adductor and abdominal muscles
- Lumbar hyperlordosis
- Reduced flexibility of the posterior chain or iliopsoas muscles

Movement Pattern Considerations

Due to the interconnectivity of the adductor, proximal hip and abdominal muscles, exercise programming should focus on both postural control and gradually loaded movements across

multiple planes in order to best prevent strength asymmetries within these muscle groups which can result from decreased and abnormal loading in the presence of adductor tendinopathy.[1]

Tendon Region-Specific Exercise Recommendations[3,6,7]

Phase #1, weeks 1 to 2 (or longer as needed) *May be performed multiple times per day*
Loading Intensity: Within pain scale of 2/10
Exercise Examples: supine adduction squeeze isometrics (bent or straight knee); 5x30 seconds

Phase #2 , weeks 2 to 8 (or longer as needed) *May be performed 3x/weekly*
Loading Intensity: Within pain scale of 4/10, utilizing heavy resistance as tolerated
Exercise Examples: standing cable resisted adduction, seated adduction machine, Copenhagens short to long lever progression; 3x10 reps all exercises, 3 sec concentric:3 sec eccentric sequencing to optimize slow resistance loading pattern

Phase #3, weeks 4 to 8 (or longer as needed) *May be performed 3x/weekly, includes continuation of phase 2 exercises*
Loading Intensity: progress movement range, volume and speed as tolerated
Exercise Examples: standing cable resisted adduction (light to moderate resistance), val slide/slide board adduction, lateral skater hops (short to long range); 3 sets of 8-10 reps, perform within a controlled range to optimize energy storage and release capacity

Phase #4, gradually phased in as progression from phase 3 exercises, includes continuation of phase 2 exercises *May be performed 3-5x/weekly, as tolerated*
Loading Intensity: progress movement range, volume and speed as tolerated to optimize energy storage loading
Exercise Examples: cutting/change of direction drills, multiplanar plyometrics; exercises to be integrated within/as sports-specific training which will influence total volume

Phase to phase progressions should be based upon the athlete's ability to demonstrate a positive response to performing the exercises outlined which considers pain levels, quality of

movement and ability to gradually increase loading. Note the context may vary from athlete to athlete based on health-history related variables.

References

1. Valent A, Frizziero A, Bressan S, Zanella E, Giannotti E, Masiero S. Insertional tendinopathy of the adductors and rectus abdominis in athletes: a review. Muscles Ligaments Tendons J. 2012;2(2):142.
2. Robertson BA, Barker PJ, Fahrer M, Schache AG. The anatomy of the pubic region revisited: implications for the pathogenesis and clinical management of chronic groin pain in athletes. Sports Med. 2009;39:225–34.
3. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
4. Morelli V, Smith V. Groin injuries in athletes. Am Fam Physician. 2001;64(8):1405–15.
5. Riley G. The pathogenesis of tendinopathy. A molecular perspective. Rheumatol. 2004;43(2):131–42.
6. Thorborg K. Current Clinical Concepts: Exercise and Load Management of Adductor Strains, Adductor Ruptures, and Long-Standing Adductor-Related Groin Pain. J Athl Train. 2023;58(7–8):589–601.
7. Hölmich P, Uhrskou P, Ulnits L, Kanstrup IL, Nielsen MB, Bjerg AM, et al. Effectiveness of active physical training as treatment for long-standing adductor-related groin pain in athletes: randomised trial. The Lancet. 1999;353(9151):439–43.

Proximal Hamstring Tendinopathy

Symptom Patterns[1]

Pain located in the buttocks region at the site of the hamstring origin/ischial tuberosity which worsens during/after squatting, running, lunging, and/or prolonged sitting

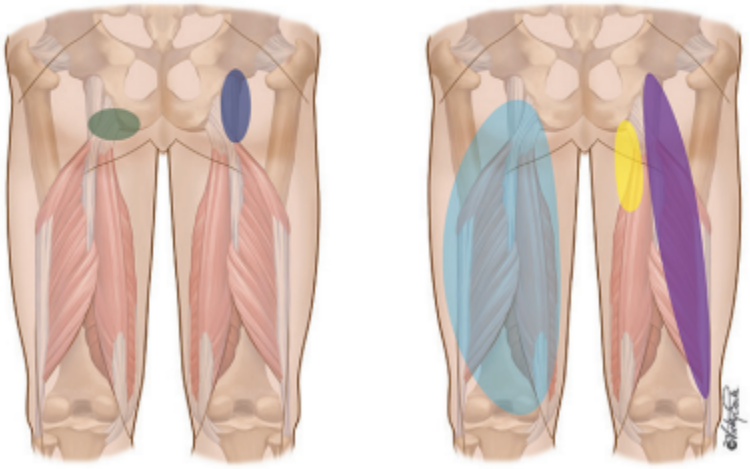

Figure. 1: Typical pain location associated with proximal hamstring tendinopathy as indicated by the green circle. Diffuse pain patterns in the hamstring region resulting from other sources are also indicated. Illustration with permission from Barça Innovation Hub 2021 Tendon Guide.[2]

Note: Consideration of differential diagnoses is important to appropriately manage proximal hamstring tendinopathy given the proximity of several complex anatomical structures in/around the buttock region. Various differential diagnoses, such as lumbar spine pathology, radiculopathy/nerve entrapment and metabolic disorders, typically have a diffuse and/or radiating pain pattern compared to the localized pattern of proximal hamstring tendinopathy.[1,2]

Causative Factors[1]

- Compression of the hamstring tendon at its attachment through repeated or sustained hip flexion
- Repeated shear forces between the ischial tuberosity and hamstring attachment from increased and repeated hip flexion angles during sport or daily activity
- Increased sport training volume/intensity within a short period of time
- Sudden inclusion of sprinting, hurdles, hill training or lunge exercise in training programs

Movement Pattern Considerations

The degree of hip flexion associated with prescribed exercise programming is an important consideration in the rehabilitation of proximal hamstring tendinopathy. High degrees of hip flexion may result in provocative tensile and compressive loads at the tendon insertion onto the ischial tuberosity[3,4] Due to this, early-stage rehabilitative programming should likely remain in low degrees of hip flexion or in hip neutral if positions loaded with hip flexion are not tolerated. For the same reasons, inclined sprints and speed training should be introduced carefully,[3,4] typically in late stages of rehab.

Tendon Region-Specific Exercise Recommendations[1]

Phase #1, weeks 1 to 2 (or longer as needed) *May be performed multiple times per day, implemented if the athlete cannot tolerate isotonics and/or loaded positions in hip flexion*
Loading Intensity: Within pain scale of 2/10
Exercise Examples: double/single leg progression bridge holds (straight leg position), prone isometric hamstring curl; 5x45 seconds

Phase #2, weeks 2 to 4 (or longer as needed) *May be performed 3x/weekly*
Loading Intensity: Within pain scale of 4/10, implementing heavy resistance where applicable avoiding deep hip flexion angles
Exercise Examples: Hip thrusters, Nordic hamstring curls, double/single leg prone hamstring curls; 2-3 sets of 8-10 reps; 3 sec concentric:3 sec eccentric sequencing to optimize slow resistance loading pattern

Phase #3, weeks 4 to 8 (or longer as needed) *May be performed 3x/weekly*
Loading Intensity: progress hip flexion angle and resistance as tolerated
Exercise Examples: double/single leg bridges, scooter pulls, sled pushes, weighted step-ups, double/single leg Romanian deadlifts, barbell squats, multi-directional lunges, leg press; 2-3 sets of 8-10 reps

Phase #4, week 8 onwards *Limit to 2 sessions/weekly, progress total sessions as tolerated*

Loading Intensity: progress movement range, volume and speed as tolerated to optimize energy storage loading
Exercise Examples: cutting/change of direction/acceleration and deceleration drills, multiplanar plyometrics, exercises to be integrated within/as sports-specific training which will influence total volume

Phase to phase progressions should be based upon the athlete's ability to demonstrate a positive response to performing the exercises outlined which considers pain levels, quality of movement and ability to gradually increase loading. Note the context may vary from athlete to athlete based on health-history related variables.

References

1. Goom TS, Malliaras P, Reiman MP, Purdam CR. Proximal hamstring tendinopathy: clinical aspects of assessment and management. J Orthop Sports Phys Ther. 2016;46(6):483–93.
2. Cook J, Rodas G, McCall A, Pruna R. Tendon Injuries in Football Players: FC Barcelona 2021 Tendon Guide. FC Barcelona; 2021. https://www.lasselempainen.fi/wp-content/uploads/2021/11/FC_BARCELONA_TENDON_GUIDE_2021-web.pdf
3. Docking S, Samiric T, Scase E, Purdam C, Cook J. Relationship between compressive loading and ECM changes in tendons. Muscles Ligaments Tendons J. 2013;3(1):7-11.
4. Soslowsky LJ, Thomopoulos S, Esmail A, et al. Rotator cuff tendinosis in an animal model: role of extrinsic and overuse factors. Ann Biomed Eng. 2002;30(8):1057-1063.

Rotator Cuff Tendinopathy

Symptom Patterns

Pain that radiates down the arm and hypersensitivity to pain upon palpation in the involved region.[1]

Causative Factors[2,3,5-13]

- Weakness of the rotator cuff, trapezius and serratus anterior muscles
- Poor control of the humeral head secondary to pain, weakness, or fatigue
- Delay in rotator cuff or scapula muscle activation which results in altered positioning of the humeral head and subacromial space
- Anterior laxity and posterior joint tightness which results in retroversion of the humeral head within the joint space
- Under or excessive rotator cuff loading
- Intrinsic factors such as aging and vascular changes
- Lifestyle factors, including obesity and smoking
- Irritation of rotator cuff tendons due to an impingement mechanism, potentially due to swelling in the supraspinatus tendon

Note: As previously mentioned, given the complex interaction of a variety of multiple factors, shoulder impingement and rotator cuff tendinopathy potentially reinforce mechanisms of each condition and are not to be assumed to occur in isolation.[11,12]

Movement Pattern Considerations

Internal and external rotation exercises during acute or subacute phases of rotator cuff tendinopathy can often be painful. A small bolster can be placed between the elbow and body to slightly abduct the arm and help slacken the supraspinatus tendon, potentially reducing irritable levels of tension.[14] With further degrees of shoulder abduction (90 degrees or more), electromyographic studies show signal increases in the supraspinatus, infraspinatus and teres minor.[15]

Tendon Region-Specific Exercise Recommendations[3,16]

Phase #1, weeks 1 to 2 (or longer as needed) *May be performed multiple times per day*
Loading Intensity: Within pain scale of 2/10 but performed in direction of pain or weakness
Exercise Examples: internal/external rotation isometric holds (low to high angles as tolerated, elbow supported, static or manual resistance); 3x30 seconds

Phase #2, weeks 2 to 4 (or longer as needed) *May be performed every other day as tolerated*
Loading Intensity: Within pain scale of 4/10, implementing light to moderate resistance performed slowly
Exercise Examples: cable or dumbbell resisted internal or external rotation (elbow supported, progress to scaption plane and 90/90 position as tolerated); 2-3 sets of 5 reps; 3 sec concentric:3 sec eccentric sequencing to optimize slow resistance loading pattern

Phase #3, weeks 4 to 8 (or longer as needed) *May be performed 3x/weekly*
Loading Intensity: progressing positioning and applied resistance as tolerated
Exercise Examples: prone/sidelying/standing manual resisted ER/IR, Diagonal and empty-can shoulder raise patterns; 2-3 sets of 8-10 reps; 2 sec concentric:5 sec eccentric sequencing to optimize eccentric loading

Phase #4, week 8 onwards *Limit to 2-3 sessions/weekly, progress total sessions as tolerated*
Loading Intensity: progress movement range, volume and speed as tolerated to optimize energy storage loading
Exercise Examples: ball throw variations, push-up plyometrics

Phase to phase progressions should be based upon the athlete's ability to demonstrate a positive response to performing the exercises outlined which considers pain levels, quality of movement and ability to gradually increase loading. Note the context may vary from athlete to athlete based on health-history related variables.

References

1. Gwilym S, Oag H, Tracey I, Carr A. Evidence that central sensitisation is present in patients with shoulder impingement syndrome and influences the outcome after surgery. J Bone Joint Surg Br. 2011;93(4):498–502.
2. Michener LA, Subasi Yesilyaprak SS, Seitz AL, Timmons MK, Walsworth MK. Supraspinatus tendon and subacromial space parameters measured on ultrasonographic imaging in subacromial impingement syndrome. Knee Surg Sports Traumatol Arthrosc. 2015;23:363–9.
3. Lewis J, McCreesh K, Roy JS, Ginn K. Rotator cuff tendinopathy: navigating the diagnosis-management conundrum. J Orthop Sports Phys Ther. 2015;45(11):923–37.
4. Myers JB, Hwang JH, Pasquale MR, Blackburn JT, Lephart SM. Rotator cuff coactivation ratios in participants with subacromial impingement syndrome. J Sci Med Sport. 2009;12(6):603–8.
5. Sein ML, Walton J, Linklater J, Appleyard R, Kirkbride B, Kuah D, et al. Shoulder pain in elite swimmers: primarily due to swim-volume-induced supraspinatus tendinopathy. Br J Sports Med. 2010;44(2):105–13.
6. Cook J, Purdam CR. Is tendon pathology a continuum? A pathology model to explain the clinical presentation of load-induced tendinopathy. Br J Sports Med. 2009;43(6):409–16.
7. Tempelhof S, Rupp S, Seil R. Age-related prevalence of rotator cuff tears in asymptomatic shoulders. J Shoulder Elbow Surg. 1999;8(4):296–9.
8. Hegedus EJ, Cook C, Brennan M, Wyland D, Garrison JC, Driesner D. Vascularity and tendon pathology in the rotator cuff: a review of literature and implications for rehabilitation and surgery. Br J Sports Med. 2010;44(12):838–47.
9. Rechardt M, Shiri R, Karppinen J, Jula A, Heliövaara M, Viikari-Juntura E. Lifestyle and metabolic factors in relation to shoulder pain and rotator cuff tendinitis: a population-based study. BMC Musculoskelet Disord. 2010;11:1–11.
10. Garving C, Jakob S, Bauer I, Nadjar R, Brunner U. Impingement syndrome of the shoulder. Deutsches Ärzteblatt International. 2017; 114(45), 765-776.
11. Lewis J. Rotator cuff tendinopathy. Br J Sports Med. 2009;43:236–241.
12. Lewis J, McCreesh K, Roy J, Ginn, K. Rotator cuff tendinopathy: navigating the diagnosis-management conundrum. J Orthop Sports Phys Ther. 2015;45(11),923-937.
13. Ishikawa H, Kurokawa D, Muraki T, Nagamoto H, Takahashi H, Yamamoto N, Izumi S. Increased external rotation related to the soft tissues is associated with pathologic internal impingement in high-school baseball players. Journal of Shoulder and Elbow Surgery. 2022; 31(9),1823-1830.
14. Williams GR, Kelley M. Management of rotator cuff and impingement injuries in the athlete. J Athl Train. 2000;35(3):300-315.

15. Edwards PK, Ebert JR, Littlewood C, Ackland T, Wang A. A Systematic Review of Electromyography Studies in Normal Shoulders to Inform Postoperative Rehabilitation Following Rotator Cuff Repair. J Orthop Sports Phys Ther. 2017;47(12):931-944.
16. Dejaco B, Habets B, van Loon C, van Grinsven S, van Cingel R. Eccentric versus conventional exercise therapy in patients with rotator cuff tendinopathy: a randomized, single-blinded, clinical trial. Knee Surg Sports Traumatol Arthrosc. 2017;25:2051–9.