The EdUReP Model for Nonsurgical Management of Tendinopathy

Tendinopathy is a common and substantial source of morbidity worldwide. Various anatomical and functional predispositions combine with abrupt changes in mechanical loading to cause characteristic histological maladaptations in tendons. The nature and latency of cellular changes in tendinopathy makes many common treatments less-than-optimal options. This Perspective presents the EdUReP model for nonsurgical management of tendinopathy, a model that considers sources of pathology at the cellular, anatomical, and functional levels. The EdUReP model addresses possible sources of symptoms at the levels of pathology, impairment, functional limitation, and disability through Educational interventions, periods of tendon Unloading and controlled Reloading, and implementation of Prevention strategies. The EdUReP model is an evidence-based treatment construct that aims to reduce functional limitation and disability through amelioration of tissue pathology. [Davenport TE, Kulig K, Matharu Y, Blanco CE. The EdUReP model for nonsurgical management of tendinopathy. Phys Ther. 2005;85:1093–1103.]

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Tendinopathy is a common musculoskeletal disorder that causes substantial annual morbidity, involving missed workdays, interpersonal and financial difficulties, and emotional distress. Current physician and physical therapist management of tendinopathy involves many treatments that lack convincing empirical support. Common nonsurgical medical management of tendinopathy involves oral nonsteroidal anti-inflammatory drugs and corticosteroid injections, although neither intervention has substantial empirical support for medium- or long-term efficacy in reducing symptoms or improving function. Rush and Shore found that rheumatologists and physiatrists place a slightly greater value on ultrasound than active exercise for patients with tendinopathy. Other passive modalities, including many with unsubstantiated efficacy, also were perceived as effective treatments. In addition, the Guide to Physical Therapist Practice lists massage and other symptom-driven interventions as accepted treatments, even though their effectiveness also is largely unsupported.

Despite the ubiquitous and disabling nature of tendinopathy, researchers are only now beginning to understand its etiology and underlying pathology. Recent advances in the scientific understanding of tendon structure and function compelled the development of the EdUReP (Education, Unloading, Reloading, Prevention) model. The EdUReP model is a theoretical framework for the clinical management of people with tendinopathy, informed by evidence from basic and clinical science. The EdUReP model is based on hypothesized relationships between the characteristic myotendinous pathology of tendinopathy and resulting impairments, functional limitations, and disabilities.

**Homeostatic Tendon Has an Organized Structure, Innervation, and Vascularization**

Tendons attach to skeletal muscle at the myotendinous junction and to bone at the teno-osseous junction. Tendon is histologically categorized as dense regular connective tissue. Like most connective tissues, tendons are relatively acellular. The acellular component of connective tissues is called “extracellular matrix” (ECM). The primary organic component of tendon ECM is the protein collagen, a fibrous inextensible ECM protein that helps maintain the structural integrity of tissues and organs throughout the body. In tendon, type I collagen organizes into fibers that are oriented parallel to each other in the direction of transduced tensile forces. Tendon fascicles are organized spirally from the myotendinous junction to the osseous attachment, contributing to the tendon’s strength during loading. Undifferentiated fibroblasts called “tenocytes” reside among the collagen fibers. Tenocytes synthesize and secrete ECM components including collagen, glycoproteins, and proteoglycans. Tendons that are frequently subjected to compressive forces, such as the human flexor digitorum profundus in the carpal tunnel, may exhibit characteristics similar to those of fibrocartilage. Layers of loose connective tissue provide tendon’s characteristic anatomical arrangement. Tendons also are commonly enveloped by synovial sheaths.

Optimal treatment for people with tendinopathy requires intervention at all levels of disablement.

Neurovascular structures of tendon are mainly located in the endotenon and epitenon. Blood supply to most tendons comes from the vasculature that supplies the attached muscle and bone by way of the myotendinous junction and teno-osseous junction, respectively.
Many tendons have characteristic “watershed zones” of hypovascularity resulting from poor overlap between osseous and muscular sources of blood. Colloid-like properties of proteoglycans in the ECM create conduits for nutrient, oxygen, and metabolic waste perfusion and diffusion between capillaries and tenocytes. Ljung and colleagues established that sympathetic axons were associated with arterioles and that primary afferent axons were associated with smaller-diameter vasculature originating in the human extensor carpi radialis brevis tendon.

Excessive Loading Causes Maladaptations in Tendon Structure That Lead to Pain
Consistent with the predictions of the Physical Stress Theory, the excessive timing, direction, and amount of myotendinous loading causes pathology. The regions proximal and distal to the myotendinous junction appear to be the most susceptible to damage and disruption during acute loading of the myotendinous unit. Animal studies have clearly shown that the region of the myotendinous unit rapidly adapts to changing load conditions. Whether local disruption of the myotendinous junction increases the potential for disruption of the tendon midsubstance has not been determined. Damage to one component of a load-bearing structure, however, will shift loads to other structural components and exacerbate failure of these structures. It also has been established that normal tendon responds to changes in mechanical stress levels to maintain homeostasis. Alterations in tendon structure resulting from changes in loading cause temporary disruption of the tendon midsubstance. Astrom and Rausings suggested that chronic overuse injuries cause microtears in the tendon midsubstance. These microtears may represent collagen fiber rupture and disruption of collagen fiber packaging within individual fascicles that weaken the tendon’s ability to resist tensile loads. In addition, excessive acute and chronic loads also may result in partial muscle tears.

A number of animal models have been used to investigate chronic overuse tendon injuries. Soslowsky and colleagues developed a model that matches human tendinopathy well because of the anatomical and functional similarities of rat and human supraspinatus tendons. In this model, rat supraspinatus tendons were excessively loaded by a downhill running task that mimics overuse syndromes involving shoulder level and overhead activities in humans. Soslowsky and colleagues showed that treadmill running with a 10% decline at constant velocity significantly decreased the maximum failure stress load of the supraspinatus tendon. Downhill running also resulted in the increased cross-sectional area and cellularity of the tendon, as well as misalignment of collagen fibers with respect to the long axis of the tendon. Both the histological and biomechanical properties of the supraspinatus tendon remained abnormal even after 16 weeks of downhill running. These data suggest that the tendon did not fully adapt to the increased functional demand of decline running and remained susceptible to further injury. Changes in force demand on a tendon may prevent tenocytes from either repairing the initial damage or optimally adapting to the new loading state. Incomplete cellular and structural adaptations initiate a spiraling cycle of decline, where compromised tendon structure may lead to further tissue damage and progressively greater functional deficits.

Arguments that symptoms of tendinopathy originate from an active inflammatory process are unconvincing because the majority of studies have failed to show inflammatory infiltrates in human biopsy samples. Rather, the symptoms of tendinopathy may be better attributed to a neurogenic origin. High levels of
tension and stress in tendons that commonly undergo chronic repetitive loading may cause stimulation of sensory fibers and regional anoxia. Smith and colleagues found evidence of local sympathetic dysfunction that may be related to the presence of pain in tennis elbow. The investigators observed an absence of normal sympathetic vasomotor response in the skin overlying the lateral epicondyle, indicating abnormal local microcirculatory control that may contribute to symptoms. These observations suggest that similar increases in sensory innervation also may be present during repetitive stress-induced tendinopathies. Anoxia is thought to trigger the activation of C fibers, ultimately causing pain in the anoxic region of tendon. Ljung and colleagues showed an imbalance between vasoconstrictor (sympathetic) and vasodilator (sensory) innervation in arterioles that also may predispose the tendon to poor perfusion and anoxia.

Regional musculoskeletal anatomy and function likely predispose human tendons to poor perfusion and anoxia, eventually leading to the symptoms of tendinopathy. One example is the supraspinatus tendon. The supraspinatus muscle passes from its broad attachments to the supraspinous fossa through a wide myotendinous junction to a narrow and flat tendon and its distal attachment at the greater tubercle of the humerus. During overhead activity, the contracted muscle tenses the tendon. The restricted space under the acromion may contribute to tendon compression, producing an impingement syndrome. The hypovascular region of the tendon also passes under the subacromial arch, causing additional predisposition for poor perfusion. Postural faults exacerbate anatomical predispositions to supraspinatus tendinopathy. People with supraspinatus tendinopathy demonstrate a characteristic clinical pattern of internal rotation of the humeral head, weakness of the external rotator and scapular stabilizer, and hypomobility of the posterior glenohumeral capsule.

This combination of factors compromises the supraspinatus tendon by decreasing subacromial volume and by allowing increased contact between bone and the tendon. In addition, these postural deficiencies alter optimal length-tension relationships for the scapular
stabilizers and upward rotators, limiting the effectiveness of dynamic scapular stabilization and further increasing force demands on the supraspinatus and other scapulohumeral muscles.

A series of maladaptations at the levels of tissue pathology and physical impairment appear to culminate in the functional limitations and disability of tendinopathy. Altered ECM composition, increased tissue cellularity, and neovascularization during the repair process can weaken the structural integrity of the tendon midsubstance. Along these lines, Nirschl previously described a 5-stage model for pathogenesis of chronic overuse tendon injuries in humans (Table). Eventually, tissue maladaptations lead to poor tissue perfusion and anoxia, which contribute to tendon pain and disability.

The EdUReP Model
The efficacy of many clinically accepted treatments for tendinopathy likely is compromised by their failure to address histopathologic sources of symptoms (Fig. 2A).

The tissue pathology characterizing tendinopathy seems more strongly correlated with functional limitations than with symptoms. Ruptured tendons, however, are significantly more degenerated than pathologic and control tendons, and clinical experience suggests that some people with tendon ruptures may not experience any prior symptoms. The potentially silent nature of histopathological changes in tendinopathy may cause affected tendons to be at risk for additional pathology even after the loading state returns to normal and initial symptoms resolve (Fig. 3). Amelioration of functional limitation and disability strictly related to pain may correspond with only a mild improvement of tissue quality (ie, restoration of Nirschl stage IV tendon pathology to stage III), leaving the person at elevated risk for recurrence of symptoms and progression of the disease. Goals of nonsurgical management should include: (1) reversing disease progression at the level of tissue pathology, (2) the person’s return to previous level of activity unlimited by symptoms or other residual physical impairments, (3) preventing disease recur-
rence, and (4) enabling the person to manage their condition independently. Correspondingly, the objective for intervention at the level of tendon pathology should be the complete restoration of tissue quality in order to reduce the likelihood of recurrent symptoms (eg, restoration of Nirschl stage IV tendon pathology to normal tissue). This approach to treatment of tendinopathy requires integrated interventions at all levels of pathology, impairment, functional limitation, and disability (Fig. 2B).

The mnemonic EdUReP emphasizes the components of Education, Unloading, Reloading, and Prevention in a model for nonsurgical management of tendinopathy. Education is the first intervention at the levels of impairment, functional limitation, and disability. To ensure optimal recovery, people with tendinopathy must recognize the etiology and pathological process of tendinopathy, as well as the nature of the contributing postural and biomechanical impairments. Education also should include a collaborative plan for the patient to self-manage pathology and symptoms. The model also features mechanical unloading and adaptive reloading of the affected tendon that intervene at the level of tissue pathology to prevent disease progression and symptom recurrence. Tendon unloading and reloading occur through behavioral change by the patient, bracing or orthoses, and interventions that address postural and biomechanical impairments. Finally, a long-term plan for preventing progression of tissue pathology and symptom recurrence must be carried out when the patient no longer has symptoms and eventually returns to premorbid activities.

**Education**

A variety of self-care, work, and sport behaviors may place people with tendinopathy at a higher risk for aggravating symptoms and speeding the progression of tendon pathology. Collaborative psychoeducational interventions that alter risky behaviors contribute to successful nonsurgical management of tendinopathy. Although a comprehensive review of the evidence regarding counseling strategies for behavioral change is beyond the scope of this Perspective, the 5 A’s construct is one approach to behavioral counseling that holds promise for facilitating behavioral change in people with tendinopathy.

The components of the 5 A’s construct are assess, advise, agree, assist, and arrange. Assessment of the patient with tendinopathy involves asking questions about behavioral health risks, as well as his or her preferred behavioral change goals, methods, and constraints. The patient’s level of knowledge related to tendinopathy and overall health literacy also may be solicited. Effective advice for people with tendinopathy includes specific and personalized behavioral change counseling based on the learning needs assessment, and should include information about the potential harms and benefits of this advice. Patients with tendinopathy and their physical therapists should agree on the goals and methods of psychoeducational interventions to alter or avoid risky behaviors. The behavioral change plan is designed in collaboration, with the patient’s decision making informed by the physical therapist’s advice. People with tendinopathy require assistance to carry out the behavioral change plan, which physical therapists may provide through clinic-based treatments and self-help components of the EdUReP model. Finally, arrangement of follow-up contacts allows for additional assistance and alteration of the behavioral change plan. Arrangement of follow-up contacts also reinforces the importance of behavioral change to the individual. Physical therapists are in a unique position to follow up with brief educational interventions because of their relatively frequent contact with their patients. Although follow-up contacts may be formal (eg, special informational classes) or informal (eg, during a regular clinic visit), the intent to discuss or
change a behavior change plan during these times should be clear to the patient.

Unloading

Other theoretical models predict the importance of unloading tissues to promote optimal improvement in pathology and resulting symptoms. Correspondingly, amelioration of tendon pathology seems to depend on a period of relative rest from chronic repetitive loading. Predisposing activities, errors in technique, and physical impairments perpetuate the pathophysiologic cycle of tendinopathy through inadequate synthesis of ECM proteins and subsequent degradation of tendon structure. As experts in clinical pathokinesiology, physical therapists are uniquely qualified to identify tendon unloading strategies using behavioral and mechanical methods. Behavioral methods of tendon unloading include psychoeducational interventions that patients can use to self-limit or modify their activities. Mechanical unloading of an affected tendon may be provided by orthoses, taping and bracing, equipment and workplace ergonomic modifications, and therapeutic activities to address contributing impairments.

Several investigators note the beneficial effects of activity adaptation and behavioral modification on symptoms and mechanisms of tendinopathy. Ilfeld reported successful treatment of recreational tennis players with short- and long-term elbow symptoms resulting from overuse using a combination of technique instruction and palliative strategies. One small subset of the sample had resolution of symptoms with stroke correction alone, further emphasizing the importance of behavioral change in tendon unloading. Gruchow and Pelletier also reported that changes in stroke technique successfully reduced symptoms and prevented recurrence of lateral epicondylitis. Forefoot landing generated lower ankle ground reaction forces in the Achilles and posterior tibial tendons than heel landing during jumping, and additional force reduction of up to 25% occurred through increases in hip and knee flexion during landing. Instructions to change lower-extremity position during landing were effective in reducing ground reaction forces at the ankle in adolescents and adults, which likely served to reduce the external force demand on the Achilles and posterior tibial tendons. These findings suggest that a collaborative plan for behavioral adaptation or modification must be carried out in order to provide tendon unloading. People with tendinopathy must vary their general workload (the sum of tendon loading during self-care, household, occupational, and recreational tasks) and daily tasks, identify and perform efficient movement patterns, and plan daily schedules to avoid sudden changes in their general workload. Behavioral tendon unloading plans involving athletes also must involve cross-training in multiple sport activities, training in appropriate sport technique, and avoiding abrupt increases in training volume.

The possible roles and functions of foot orthoses to address lower-extremity pain have been discussed. Acquired flat-foot deformities, leg length discrepancies, and dynamic factors (eg, excessive pronation and limited impact attenuation) may increase stress on lower-extremity tendons and increase the risk of tendinopathy. Foot orthoses have been demonstrated to reduce lower-extremity symptoms, possibly by amelioration of various biomechanical impairments. Heel lifts also have been demonstrated to decrease Achilles tendon pain. Heel lifts place the ankle in relative plantar flexion during the terminal stance of walking and running, possibly reducing the magnitude of the external dorsiflexion moment and consequent tension on the Achilles and tibialis posterior tendons.

Several studies have documented the efficacy of taping to reduce pain in affected tendons by restraining movement, providing a tactile stimulus to remind the person to decrease the magnitudes of aggravating movements, or improving loading mechanics in order to decrease pain. For example, wrist splints or taping that limit wrist and finger movement would appear to unload the extensor carpi radialis brevis and extensor digitorum tendons by decreasing the muscle activity needed to stabilize the wrist and fingers during sports or during grasping activities.

Manual and exercise interventions to reduce contributing impairments in range of motion, muscle performance, and motor control also provide mechanical unloading for affected tendons. For example, the tibialis posterior musculotendinous unit restrains foot pronation in closed chain activities because of its complex insertion sites throughout the plantar midfoot and proximal forefoot. An effective mechanical unloading strategy for the tibialis posterior tendon to ameliorate posterior talocrural joint dorsiflexion might limit the magnitude of foot pronation during closed-chain activities. Clinical studies and anatomical studies suggest that ankle dorsiflexion is a composite motion involving both talocrural joint dorsiflexion and foot pronation. Therefore, excessive foot pronation may be related to limited talocrural joint dorsiflexion range of motion or limited eccentric control of the calf. Interventions to improve the contribution of talocrural joint mobilization and calf soft tissue mobilization, stretching, and strengthening.
Reloading

The Physical Stress Theory predicts that a period of controlled reloading is needed for amelioration of impairment, functional limitation, and disability related to tendinopathy without progression of underlying pathology. The optimal length of this reloading period has not been established empirically. The length of intervention periods in clinical studies of eccentric exercise as a reloading strategy suggest that people with tendinopathy achieve good clinical results and amelioration of tissue pathology in approximately 12 weeks. Individual differences in disablement, however, suggest that a broader range of appropriate times for the reloading period is needed. Advanced tendon pathology should be assumed in all patients with tendinopathy because of the correlation between function and tissue degeneration. Patients should be monitored for acute increases in functional limitation or disability that characterize maladaptive reloading and pathological progression.

Appropriate reloading stress on the affected tendon may be induced by behavioral and mechanical methods. Behavioral tendon reloading involves psychoeducational interventions that allow the patient to modify the volume and technique of an activity to promote reloading. Mechanical reloading also must occur in a gradual and stepwise manner. Use of body weight–supported environments (eg, treadmill apparatus and swimming pools) and weaning from unloading devices (eg, as braces and orthoses) are other examples of mechanical reloading strategies. These interventions seem worthy of future study in people with tendinopathy.

Effective reloading programs also include eccentric exercise. Recent research studies documented a high rate of return to premorbid activities in athletes with Achilles tendinopathy who participated in nonsurgical programs involving eccentric exercise. Eccentric reloading also resulted in elimination of focal thickening of affected Achilles tendons and decreased hypoechoicity (improved collagen fiber organization); other findings that characterize Achilles tendon histopathology were completely reversed in 73% of people participating in a controlled eccentric reloading program. The mechanism for improvement in tissue pathology and symptoms remain unclear. Despite resolution of symptoms in 100% of people with Achilles tendinopathy after an eccentric reloading program, levels of intratendinous glutamate—one measure of neurogenic inflammation—were not significantly different before or after participation. However, sclerosing injections into regions of tendon neovascularization in people with tendinopathy eliminated both neovascularization and symptoms, which seems reasonable given that the injections also may have obliterated sensory (nociceptive) innervation associated with small-diameter vasculature. Currently, research has been documenting more beneficial effects than adverse effects of eccentric exercise as a treatment for patients with tendinopathy. Although additional studies are needed to document possible adverse effects, eccentric reloading should be initiated and progressed at slow speeds and sufficient loads to prevent the progression of tendon pathology. The most widely studied eccentric reloading program was created by Alfredson and colleagues. This program involves the recommendation that mild tendon symptoms during eccentric reloading are acceptable. The safety of this protocol is supported by the absence of tendon ruptures documented resulting from this approach. However, clinicians using a similar training paradigm should monitor patients for increases in functional limitation and disability that may indicate progression of pathology, because ultrasonographic findings of pathology are significantly correlated with function rather than symptoms. Clinicians also should avoid prescribing abrupt changes in training speed and volume, because these training errors are consistent with the etiology of many overuse syndromes and may likely worsen existing pathology.

Prevention

The chronic nature of symptoms, functional limitations, and disability related to a progressive underlying pathology in tendinopathy emphasizes the importance of prevention in a comprehensive model of nonsurgical management. Optimal secondary prevention programs for people with tendinopathy have yet to be determined. In the EdUReP model, prevention may be viewed as a targeted continuation of prior education, unloading, and reloading phases, with special emphasis on the patient’s independence in self-management of residual symptoms and physical impairments after a pain-free return to previous activities. In the late phases of rehabilitation, patients with tendinopathy no longer report symptoms that initially allowed for rudimentary self-monitoring of pathologic progression, making prevention strategies particularly important. Prevention strategies may include periodic follow-up after discharge from the formal physical therapy program to ensure the person’s consistent adherence to the prevention plan.

Summary

This Perspective presented a model for the nonsurgical management of tendinopathy based on present understanding of its cellular, anatomical, and functional sources of pathology. The acronym EdUReP emphasizes the model’s components of Education, periods of mechanical Unloading and controlled ReLoading of the affected tendon, and implementation of a plan for Prevention of disease progression and symptom recurrence. The EdUReP model is an evidence-based treat-
ment construct that aims to reduce functional limitation and disability through amelioration of tissue pathology.

References
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