

CURRENT CONCEPTS OF MUSCLE AND TENDON ADAPTATION TO STRENGTH AND CONDITIONING

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ABSTRACT

Injuries to the muscle and/or associated tendon(s) are common clinical entities treated by sports physical therapists and other rehabilitation professionals. Therapeutic exercise is a primary treatment modality for muscle and/or tendon injuries; however, the therapeutic exercise strategies should not be applied in a “one-size-fits-all approach”. To optimize an athlete’s rehabilitation or performance, one must be able to construct resistance training programs accounting for the type of injury, the stage of healing, the functional and architectural requirements for the muscle and tendon, and the long-term goals for that patient. The purpose of this clinical commentary is to review the muscular and tendinous adaptations associated with strength training, link training adaptations and resistance training principles for the athlete recovering from an injury, and illustrate the application of evidence-based resistance training for patients with a tendinopathy.

Key Words: Muscle, resistance training, therapeutic exercise, tendon

Level of Evidence: 5

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INTRODUCTION

Sports physical therapists frequently prescribe therapeutic exercises (TE) in order to facilitate an athlete's recovery after acute injury or surgery.¹⁻⁴ TE strategies should not be applied in a "one-size-fits-all approach". To optimize an athlete's rehabilitation or performance, one must be able to construct resistance training programs (or apply an evidence-based TE program when available) accounting for the type of injury, the stage of healing, the functional and architectural requirements for the muscle and tendon, and the long-term goals for that patient. Increasing muscular strength, muscular endurance, and muscular power is at the core of improving health and reducing the risk of injury in healthy athletes and athletic individuals.¹⁻⁴

In order to optimize an athlete's rehabilitation or performance, a physical therapist must be able to construct resistance training programs applying evidence-based principles of exercise prescription. The purpose of this clinical commentary is to present the muscular and tendinous adaptations associated with strength training, link training adaptations and resistance training principles for the athlete recovering from an injury, and illustrate the application of evidence-based resistance training for a patient with a tendinopathy.

MUSCULAR AND TENDINOUS ADAPTATION TO RESISTANCE TRAINING

Performing resistance training exercises will cause muscular and tendinous adaptations in patients and healthy athletes.^{2, 5-16} These training adaptations can be advantageous for helping an athlete recover from an injury or surgery and/or to improve aspects of athletic performance. Increases in strength associated with prolonged resistance training occur due to a combination of neural and morphologic adaptations.^{2,17-22} The focus of this commentary will be on the morphologic adaptations of the muscle and tendon; however, it is important for physical therapists to appreciate that immediate gains in strength may be the result of neural adaptations.^{2,17-22}

Physiological Cross-Sectional Area (PCSA)

A key morphologic adaptation associated with increases in strength due to resistance training is the increase in physiological cross-sectional area (PCSA) of skeletal muscle. Increases in PCSA can be observed

via diagnostic imaging including magnetic resonance imaging [MRI], diagnostic ultrasound, and computerized tomography, as early as two months after initiating a training program.^{2,20-24} MRI is considered the gold standard for measuring changes in PCSA.²³ Diagnostic imaging is routinely utilized in research to assess changes in PCSA; however, utilization of imaging techniques is financially prohibitive in most clinical situations. In addition, strength gains experienced by patients during the early phases of a rehabilitation program are likely the result of neural adaptations with subsequent strength gains associated with muscular hypertrophy occurring one to two months after initiating training.^{1,2,21,22} Physical therapists, and other rehabilitation clinicians, should instead assess force production with traditional clinical tests (e.g., manual muscle testing, dynamometry) or with functional performance tests. A measure of a muscle's cross-sectional area could be determined with a girth measurement using a tape measure; however, this anthropometric measure can only provide a comparison to the uninjured side (e.g., thigh, calf, upper arm, chest) and cannot provide a measure of an individual muscle nor provide the clinician with an indication of either strength or functional performance.

Increases in PCSA plateau at some point between six months to one-year after initiating a training program.^{2,23} Muscular hypertrophy occurs in both genders; however, muscular hypertrophy in women is typically lower. Strength gains and increases in cross-sectional area in females occur at 80% of those seen in their male counterparts.²⁵⁻²⁷ Lower circulatory androgens and androgen receptor content in females may explain the lower propensity for increases in PCSA as compared to that seen in males.²⁸ PCSA decreases across the lifespan; however, aging adults who perform resistance training exercises can experience an increase in PCSA. Fiatarone et al found PCSA increases in the thigh in nonagenarians after 12 weeks of resistance training.²⁹ The potential to experience increases in PCSA, and thus increases in muscular strength, may help to reduce risk of musculoskeletal injury and/or improve function.²⁹⁻³¹

Hypertrophy

The muscle fiber, or myofibril, is comprised of thousands of sarcomeres. The sarcomere, which is the functional unit of the muscle, consists of several

proteins that are involved in the contraction of skeletal muscles. Hypertrophy of muscle fibers is considered the primary mechanism associated with increases in PCSA due to resistance training.^{2,24} During resistance training, microdamage to the architecture of the muscle occurs. This “damage” to the muscle stimulates activity and proliferation of satellite cells.³²⁻³⁷ Satellite cells, precursors to myofibers, are located between the sarcolemma and the basal lamina.^{38,39} Satellite cells can contribute their nuclei to a myofibril; this addition of the nuclei can aid synthesis of contractile proteins.⁴⁰ Addition of new actin and myosin contractile proteins increases the PCSA of the muscle. Satellite cells may also fuse existing cells to each other to create new myofibrils.⁴¹

A proposed second mode of muscular hypertrophy is known as sarcoplasmic hypertrophy.^{42,43} Sarcoplasmic hypertrophy is the result of increases in fluid and noncontractile components that comprise muscle.^{42,43} This form of hypertrophy, which has been observed in bodybuilders, may be the result of a specific training stimulus.^{44,45} Researchers have found that bodybuilders have higher glycogen content levels than an individuals who primarily perform exercises to enhance muscular power.^{44,45} Differences in muscle bulk due to changes in noncontractile elements of the muscle in bodybuilders may be due training strategies. For example, bodybuilders may perform 6 to 12 repetitions per set with training loads ranging from 67 to 85 percent of their 1-repetition max (RM) lift whereas recommended training volumes and loads to enhance power are one to three repetitions performed at 75 percent or higher of one’s 1-RM.²

Resistance training appears to have a greater effect on increasing PCSA in the muscles of the upper extremity than those in the lower extremity.⁴⁶⁻⁴⁹ There are two potential explanations for this observation. First, the muscles of the lower extremities (LE) may experience a lower level of PCSA gains in response to a new resistance training program because many of the LE muscles are consistently under load in weight-bearing positions.⁴⁷ Additionally, the loads during resistance training of the LE may not be high enough to cause increases in PCSA.⁴⁷ Second, the upper extremity muscles have higher concentrations of androgen receptors.⁴⁹

Researchers have observed, via biopsy studies of the UE and LE muscles, increases in fiber area with both Type I and Type II fibers experiencing hypertrophy in response to resistance training.⁵⁰⁻⁵² Initial adaptations to resistance training (during the first one to two months after initiating a program) occur due to hypertrophy primarily of Type II fibers.² Of potential clinical interest to physical therapists is the finding that Type II fibers, in injured individuals or in those who have reduced or altogether stopped their training program, atrophy quicker than Type I fibers.² Type I muscle fibers also hypertrophy in response to resistance training programs; however, increases in fiber size are usually observed in studies when researchers perform assessments 2 or more months after initiation of a training program.^{53,54}

Given the right stimulus, most characteristics of the musculotendinous unit including fiber type, fiber length and diameter; tendon length, distribution, and architecture; and the glycolytic/oxidative pathways change.⁵⁵ It is well established that muscle fiber transformation can take place with specialized training stimuli.⁵⁵ For example, regardless of muscle or species (both animal and human), many researchers have demonstrated muscle fiber transformation from Type II (fast twitch) to Type I (slow twitch).^{56,57} This fast-to-slow fiber transformation is generally described as a complete change of the fast twitch fiber both histochemically and biochemically without any actual differences in the slow twitch fibers that did not convert. Additionally, the process of transformation is not merely a loss of fast twitch fibers but a change in the amount of metabolic enzymes and sarcoplasmic reticulum available for use and interestingly, both adapt much more easily than the contractile proteins.⁵⁵ Resistance training may cause ruptures of the Z-disk followed by subsequent longitudinal division of the muscle fiber. This response has been observed in animal studies,⁵⁸⁻⁶⁰ and is postulated to also occur in humans. Training also leads to increases in the size of myofibrils as well as increases in myosin filaments on the periphery of each fibril.²

Hyperplasia, which is a term describing an increase in the total number of muscle fibers, has also been proposed as a potential mechanism associated with increases in muscular size. However, evidence to

support this mechanism is lacking. Current thought is any contribution to PCSA via hyperplasia is minimal if at all.^{2,61,62}

Tendinous Adaptations

Increases in tendon stiffness in response to resistance training have been identified in both animal and human studies.⁶³⁻⁶⁶ Stiffness describes a mechanical property of the tendon. Stiffness is the force required to stretch a tendon per a unit of distance. Increased stiffness can impact the ability of the muscle to rapidly generate force. In addition, tendons respond to chronic resistance training by increasing total number of collagen fibrils, increasing the diameter of collagen fibrils, and increasing in fibril packing density.⁶⁷⁻⁷¹

GENERAL CELLULAR EVENTS ASSOCIATED WITH AN INJURY OF A MUSCLE AND TENDON

After a soft tissue injury to a muscle and/or tendon, a series of cellular events occur as part of the healing process (Table 1).⁷² Appreciating the events that

occur during the healing process should influence the type of exercises prescribed by the rehabilitation professional.

The Acute Stage

Onset of an inflammatory response occurs the moment of an acute injury. The function of the inflammatory response is to halt of the progression of cellular injury, initiate the body's healing process, and to protect the area from further injury.⁷² The onset of an acute injury is marked by the classic inflammatory signs: rubor (redness), tumor (swelling), calor (warmth or heat), and dolor (pain).⁷² The swelling associated with the acute inflammatory process is a result of spread of exudate (the plasma and serum proteins) from the damaged vessels in the region of injury. The function of the exudate is to initiate repair at the site of the injury. A negative consequence associated with swelling is the onset and/or increase pain due to the pressure applied to free nerve endings.⁷²

The cellular events associated with the acute injury stage can last up to six days. Appropriate treatment

Table 1. General Cellular Events Associated with a Muscle and/or Tendon Injury⁷²

Time Frame After Injury	Cellular Events
First 48 hours	<ul style="list-style-type: none"> • Initial vasoconstriction and coagulation upon rupture of blood and lymphatic vessels • Immediate swelling at injury site caused by cellular components (e.g., exudate) leaking from the blood and lymphatic vessels leaking into wound area • Clot formation • Hallmark signs associated with an acute injury: redness, warmth, pain, and swelling • Chemical mediators attracted to the site to control the inflammatory process • Vasodilation occurs shortly thereafter the initial injury • Phagocytosis (removal injured/dead cellular material)
Day 2 to 4	<ul style="list-style-type: none"> • Reduction in inflammation • Reabsorption of clot • Increase in granulation tissue (fibroblasts, myofibroblasts, and capillaries) • Angiogenesis
Day 4 to 21	<ul style="list-style-type: none"> • Scar formation • Production of collagen, elastin, and ground substance • Growth of scar ceases at the end of the phase
Day 21 to 60	<ul style="list-style-type: none"> • Scar remodeling • Collagen thickens and strengthens

prescription is key to facilitating the progression of healing from the acute to subacute stages.⁷² Delaying treatment may fail to modulate a patient's pain experience whereas aggressive treatment may increase a patient's pain experience or further damage the injured region. Appropriate forms of treatment for an acutely injured region include protection or rest, modalities (e.g., cryotherapy), gentle range of motion exercises, and possibly gentle isometric strengthening exercises (Table 2).⁷²

The Subacute Phase

The subacute phase of healing begins as early as the third day after trauma/injury and lasts for up to 21 days. New capillary growth, known as angiogenesis, occurs in the injured area early in this stage.⁷² Also, fibroblasts synthesize new collagen to replace the damaged tissue. Collagen is the primary structural protein of soft tissue structures. This newly formed collagen is weak and thin and is oriented haphazardly.⁷² It is clinically relevant to appreciate the structural integrity of the newly repaired collagen tissue. Aggressive or unsupervised treatments may damage this structurally weak tissue and delay healing. Common practice during the early part of the subacute phase is to have a patient continue range of motion exercises in order to restore any remaining deficits, initiate gentle, pain-free stretching, and initiate either isometric or low-load, high-repetition

exercises. A key to successful rehabilitation is to not have the patient perform exercises that provoke or increase pain (*Note: there are instance when a patient should experience an increase in pain during exercise. Specifically, this relates to rehabilitation of tendinosis conditions*). For example, a clinician may prescribe two sets of 15 repetitions of a short arc quad exercise for a patient with anterior knee pain. The exercise should be terminated if the patient begins to experience symptoms during the performance of the repetitions (the clinician would document how many sets and reps that the patient was able to perform and how much weight was utilized).

The Chronic Stage (Maturation and Remodeling Stage)

The term chronic is sometimes used in reference to the final stage of healing (e.g., the maturation and remodeling stage) or as a descriptor of one's medical condition state (e.g., chronic low back pain or chronic pain due to arthritis). The chronic stage of healing is the final stage of healing that begins around day 21 and continues up to 12 months after injury.⁷² During this stage of healing collagen is remodeled in response to applied forces. Progression of the exercise program will increase tensile strength of the tissue. Patients are typically able to initiate functional strengthening (e.g., squats, lunges) followed by plyometric or power training exercises.^{1,2}

Table 2. Summary of Healing Response and Appropriate Conservative Rehabilitation Treatments

The Acute Stage (Inflammatory response) 1-6 days post-injury	The Subacute Stage (Repair and Healing Phase) Begins as early as day 3	Chronic Stage (Maturation and Remodeling Stage) Begins about 3 weeks post- injury
Exudate (plasma and serum pr-) leaks out of damaged vessels into surrounding tissue They function to repair damaged site, but can cause significant swelling Swelling → stress to free nerve endings → pain	Fibroblast activity: synthesize new collagen, the collagen is formed and oriented in a haphazard fashion. Collagen is structurally thin and weak Overstressing/aggressive therapy may reinjure tissue Not stressing tissue may allow adhesions to form	Collagen is remodeled along lines of applied stress Applying appropriate volume of stress to healing tissue will help to facilitate functional orientation of collagen and increase tensile strength capability
Treatments: Modalities, gentle ROM, isometric strengthening	Treatments: initiate strengthening, continue ROM progression	Treatments: strengthening, functional strengthening, progress to plyometrics or power training as appropriate

**PATHOANATOMICAL CHANGES
ASSOCIATED WITH SKELETAL MUSCLE
AND TENDON INJURY**

Muscle Injury

Features of a strain injury of a muscle include tearing of the fibers, muscular weakness, functional loss, swelling, and pain. Strain injuries are classified as 1st, 2nd, or 3rd degree strains (Table 3). First degree strains are minor injuries associated with tearing of less than one-half of the fibers, minor pain and weakness, little to no loss of function, and little to no swelling. Second degree strains are associated with tearing of approximately one-half of the fibers, moderate pain and muscular weakness, a moderate degree of functional loss, and visible swelling. A third degree injury is associated with a complete tear, or rupture, of the muscle or tendon, significant loss of function, significant muscular weakness, and swelling. An individual who has experienced a third-degree strain may not experience pain if an innervating sensory nerve has been damaged.

Often, injury results in immobilization and it may have an unpredictable effect on muscle size, length, and strength. Position of immobilization affects sarcomeres. When a muscle is immobilized at a longer length it will experience an increase in its number of sarcomeres in series whereas immobilization of a

muscle in a shorter length will cause a decrease in the number of sarcomeres in series.⁴¹ Researchers have found that not all muscles respond in a similar fashion to immobilization. For example, when the quadriceps was immobilized for 10 weeks, the PCSA of rectus femoris was least affected, and conversely, the PCSA of the vastus medialis was affected the most.^{55,73,74} Fiber type alone does not help explain some of these differences in atrophy. The vastus medialis (VM) has a similar percent of Type I (slow twitch) fibers as the rectus femoris and yet atrophied more. Similarly, the VM's Type II fibers atrophied more than those Type II fibers of the vastus lateralis.^{55,73,74} Most muscles are comprised of similar levels of Type I and Type II fibers with some variation among studies.⁷⁵⁻⁷⁷ In general, there is either a 40%-60% split (Type I:II) or 60%-40% split (Type I:II).⁷⁸ Muscles with a higher percentage of Type I fibers have slower twitch characteristics as well as a decreased capacity for force generation, function as postural stabilization muscles, and function predominantly in endurance-type activities.^{2,55} Conversely, muscles with a higher concentration of Type II fibers have more fast twitch characteristics, are able to generate greater amounts of force, and function predominantly in shorter burst activities.^{2,55} With specific endurance training, it is possible to increase the percentage of Type I fibers.⁷⁹⁻⁸¹ However, it is not

Table 3. *Pathoanatomical Changes Associated with Muscle and Tendon Injury: 1st, 2nd, and 3rd Degree Strains*

	1 st Degree	2 nd Degree	3 rd Degree
Degree of Muscle Damage	Tear of a few muscle & tendon fibers	Tear of approximately half of the muscle fibers	Tear (rupture) of the entire muscle
Muscle Weakness	Minor	Moderate (significant)	Major
Loss of Function	None to minor	Moderate	Major
Pain	Minor	Moderate to Major	None (A lack of pain associated with 3 rd degree strain because the nerve is often significantly damaged during the injury)
Swelling	Minimal to none	Noticeable degree of Swelling	Significant degree of Swelling

Table 4. Muscle Groups and Type I Fiber Contribution

Muscle	Type I Contribution ⁸²⁻⁸⁴
Rotator Cuff (Supraspinatus, Infraspinatus, Teres Minor, Subscapularis)	Type I: 44% range 38% in subscapularis – 54% in supraspinatus)
Deltoid	Type I: 47 – 74%
Gluteus Maximus	Type I: 52-60%
Rectus Femoris	Type I: 30 – <50%
Vastus Lateralis, Medialis, Intermedius	Type I: 44 – 64%
Hamstrings (Semitendinosus, Semimembranosus, Biceps Femoris)	Type I: 44 – 67%
Gastrocnemius	Type I: 44-65%
Soleus	Type I: ~70%
Multifidus	Type I: ~65%

Table 5. General Training Guidelines for Selective Hypertrophy of Muscle Fibers

Muscle Fiber Type	Training Program
Type I Muscle Fibers	Low resistance, volume, and aerobic exercise
Type II Muscle Fibers	High intensity / low volume training programs (e.g., power training)

clear if training causes an increase in the percentage Type II fibers in strength and power athletes.⁸⁰ Table 4 lists a few of the major muscles and their Type I fiber proportions.

As previously mentioned, the long held belief about the specificity of training is true. As an example, endurance training signals the satellite cells to produce Type I muscle fibers. Since some muscle fiber conversion (Type II-to-Type I) takes place during training, rehabilitation and training experts need to keep this in mind.⁵⁵ For example, excessive endurance training for a sprinter may result in a fast-to-slow conversion of muscle fibers. Similarly, too many repetitions for a strength and power athlete may also result in muscle type conversion from fast-to-slow. Table 5 provides general training guidelines for muscles based on fiber type.

Tendon Injury

Tendons are fibrous connective tissues that are anatomically oriented between muscle and bone. The tendon helps to facilitate joint movement and stability via the tension generated by the muscle. Tendons also store energy that may be used for later movement. For example, the Achilles tendon may store as much as 34% of the total ankle power.⁸⁵

Tendons consist of fibroblasts and an extracellular matrix. The fibroblasts synthesize the components of the extracellular matrix including the ground

substance, collagen fibers, and elastin.⁸⁶ The collagen fibers are key to providing tensile strength to the tendon. The parallel arrangement of the collagen fibers provides strength to the tendon allowing it to experience large tensile forces without sustaining injury.⁵⁵ When a tendon experiences strain levels that overload the tissue’s tensile capabilities microtrauma or macrotrauma will result.⁵⁵ Typically, strength training rarely leads to a tendon injury. However, in a diseased state, a tendon is at risk of injury or reinjury if the tendon is overloaded.⁵⁵ Clinicians are advised to prescribe training loads for patients with a tendon injury (or for that matter any musculoskeletal injury) that do not reproduce symptoms.

Tendons are largely comprised of Type I collagen and once lengthened provide a high degree of tensile strength.⁵⁵ They also act parallel with the viscoelastic component of the muscle storing energy for later use. Significant variations exist in the stiffness properties of tendons and this stiffness can affect the force-velocity characteristics of a muscle. As an example, if a tendon is too compliant (lacks stiffness) it will result in a reduced ability of the muscle to generate force. The property of a tendon is dependent on its stress and strain characteristics where stress is defined as force divided by cross-sectional area and strain is defined as the change in length divided by its resting length. Young’s Modulus helps describe the relationship between stress and

Table 6. *Tendon Injury Classifications*

Injury Classification	Description
Tendinitis	Acute, inflammatory injury Examples Peritenonitis – inflammation of the peritenon Tenosynovitis – inflammation of the synovial membrane Tenovaginitis – inflamed, thickened tendon sheath
Tendinosis	Chronic, degenerative injury not associated with an inflammatory process. Degenerative changes result from microtrauma or age.

strain. For example, a stiff tendon can accept high loads (stress) and experience very low deformation (strain). Some evidence exists suggesting that tendon stiffness and hypertrophy increases following resistance training.^{66,69,87,88}

Tendons are at risk for overuse, traumatic, and degenerative injury.^{86,89,90} Because of the anatomical relationship between the tendon and skeletal muscle, sometimes both structures are injured simultaneously. However, there are a number of injuries that are specific to the tendon itself (Table 6). It is important for clinicians to distinguish the difference between an acute, inflammatory tendon injury and a chronic, degenerative injury. Acute injuries of the tendon include peritenonitis, tenosynovitis, and tenovaginitis (Table 6). Therapists will be unable to clinically distinguish between the different types of an acute tendon injury; diagnosis would require medical studies not available to PTs. Clinical diagnosis of a tendinosis may also pose challenges for a PT. A patient who presents with a tendinosis may report experiencing pain for a prolonged period of time and not be able to describe a specific mechanism of injury. For example in a patient with a suspected Achilles tendinosis (see Appendix), the therapist must use their clinical judgment to determine if inflammation is present. Although the symptoms may be similar, palpation may reveal whether the enlarged area has inflammatory signs including redness and swelling. Another differentiating test is range of motion. If swelling is a result of inflammation, the swelling will not move with the tendon, whereas an enlarged or thickened region in the tendon will move as the tendon moves in a patient with a tendinosis. Results from rehabilitation studies suggest that individuals with a tendinosis (chronic, degenerative) benefit from intense, eccentric exercise based programs whereas

those with a tendinitis (acute, overuse) benefit from a gradual, conservative therapeutic program.⁹⁰

Case Example: Therapeutic Exercise for Achilles Tendinosis

Although skeletal muscle may be one of the most adaptable materials in the body, if the cumulative mechanical or metabolic loads on the muscle fiber are too high, the tissue will “break”. Simply stated, injury is a result of the body’s failure to adapt and muscle overtraining continues to be the number one reason for injury.

Documented injury rate among recreational runners range between 25-65%.⁹¹⁻⁹³ Midportion Achilles tendinopathy is among the most common injuries, with the incidence in recreational and elite runners ranging between 6-18%.^{89,90,94} The challenge is that up to 29% of patients with Achilles tendinopathy do not respond to conservative treatment and may require surgical intervention.^{95,96} Interestingly, as high as 45% of non-athletic patients' with Achilles tendinopathy may not respond to current eccentric protocols.⁹⁶ As stated above, tendinosis is a chronic degeneration and is associated with a conversion of Type I and a higher percentage of Type III collagen fibers resulting in a structurally altered tendon. There are no inflammatory markers and the tendon often demonstrates neovascularization. The overwhelming current evidence-based conservative treatment for Achilles tendinosis that shows favorable results in many randomized controlled studies and in systematic reviews, is an eccentric training program.^{90,97-99} Although the exact mechanism by which eccentric exercise affects tendon is unclear it is thought that the eccentric exercise encourages tissue repair and remodeling.¹⁰⁰ Recently, van der Plas et al reported that 46 patients with Achilles tendinopathy at the five-year follow-up

demonstrated significant increases in the VISA-A but with continued minimal pain.⁹⁹ Additionally, Beyer et al recently published article on the benefits of a heavy slow resistance training was evaluated and compared with current eccentric protocols and demonstrated positive results.¹⁰¹

CONCLUSION

This commentary has reviewed morphologic changes in the human muscle and tendon associated with resistance training. Sports physical therapists prescribe therapeutic exercises in order to address deficits associated with injury as well as design and implement training programs to reduce the risk of injury and to enhance sports performance. When possible, sports physical therapists should utilize evidence-based programs in the rehabilitation of musculoskeletal sports injuries. The provided case example illustrates the application of the concepts in this commentary applied to treatment for Achilles tendinosis.

REFERENCES

1. Kisner C, Colby LA. *Therapeutic Exercise: Foundations and Techniques (Therapeutic Exercise: Foundations and Techniques)*. 6th ed. Philadelphia, PA: FA Davis; 2012.
2. Baechle TR, Earle RW, National Strength and Conditioning Association. *Essentials of Strength Training and Conditioning*. 3rd ed. Champaign, IL: Human Kinetics; 2008.
3. Brotzman SB, Manske RC. *Clinical Orthopaedic Rehabilitation: An Evidence-Based Approach*. 3rd ed. Philadelphia, PA: Mosby Elsevier; 2011.
4. Manske RC. *Postsurgical Orthopedic Sports Rehabilitation: Knee & Shoulder*. St. Louis, MO: Mosby; 2006.
5. Borde R, Hortobagyi T, Granacher U. Does-response relationships of resistance training in healthy old adults: a systematic review and meta-analysis. *Sports Med*. 2015; Sep 29 [Epub ahead of print].
6. Cheema BS, Chan D, Fahey P, et al. Effect of progressive resistance training on measures of skeletal muscle hypertrophy, muscular strength and health related quality of life in patients with chronic kidney disease: a systematic review and meta-analysis. *Sports Med*. 2014; 44: 1125-1138.
7. Stewart VH, Saunders DH, Greig CA. Responsiveness of muscle size and strength to physical training in very elderly people: a systematic review. *Scand J Med Sci Sports*. 2014; 24: e1-e10.
8. Roig M, O'Brien K, Kirk G, et al. The effects of eccentric versus concentric resistance training on muscle strength and mass in healthy adults: a systematic review with meta-analysis. *Br J Sports Med*. 2009; 43: 556-568.
9. Garfinkel S, Cafarelli E. Relative changes in maximal force, emg, and muscle cross-sectional area after isometric training. *Med Sci Sports Exerc*. 1992; 24: 1220-1227.
10. Housch DJ, Housch TJ, Johnson GO, et al. Hypertrophic response to unilateral concentric isokinetic resistance training. *J Appl Physiol*. 1992; 73: 65-70.
11. Cadore EL, Casas-Herrero A, Zambom-Ferraresi F, et al. Multicomponent exercises including muscle power training enhance muscle mass, power output, and functional outcomes in institutionalized frail nonagenarians. *Age*. 2014; 36: 773-785.
12. Scanlon TC, Fragala MS, Stout JR, et al. Muscle architecture and strength: adaptations to short-term resistance training in older adults. *Muscle Nerve*. 2014; 49: 584-592.
13. Pyka G, Lindenberger E, Charette S, et al. Muscle strength and fiber adaptations to a year-long resistance training program in elderly men and women. *J Gerontol*. 1994; 49: M22-M27.
14. Vissing K, Brink M, Lonbro S, et al. Muscle adaptations to plyometric vs. resistance training in untrained young men. *J Strength Cond Res*. 2008; 22: 1799-1810.
15. Aagaard P, Andersen J, Dyhre-Poulsen P, et al. A mechanism for increased contractile strength of human pennate muscle in response to strength training: changes in muscle architecture. *J Physiol*. 2001; 534: 613-623.
16. Abe T, DeHoyos D, Pollock M, et al. Time course for strength and muscle thickness changes following upper and lower body resistance training in men and women. *Eur J Appl Physiol*. 2000; 81: 174-180.
17. Folland JP, Williams AG. The adaptations to strength training: morphological and neurological contributions to increased strength. *Sports Med*. 2007; 37: 145-168.
18. Sale DG. Neural adaptation in strength and power training. In: Jones NL, McCartney N, McComas AJ, ed. *Human Muscle Power*. Champaign, IL: Human Kinetics; 1986: 289-307.
19. Sale DG. Neural adaptations to strength training. In: Komi PV, ed. *The Encyclopedia of Sports Medicine: Strength and Power in Sport*. 2nd ed. Malden, MA: Blackwell Scientific; 2003: 281-314.

20. Ogasawara R, Yasuda T, Ishii N, Abe T. Comparison of muscle hypertrophy following 6-month of continuous and periodic strength training. *Eur J Appl Physiol.* 2013; 113: 975-985.
21. Farup J, Kjolhede T, Sorensen H, et al. Muscle morphological and strength adaptations to endurance vs. resistance training. *J Strength Cond Res.* 2012; 26: 398-407.
22. Ahtiainen JP, Pakarinen A, Alen M, et al. Muscle hypertrophy, hormonal adaptations and strength development during strength training in strength-trained and untrained men. *Eur J Appl Physiol.* 2003; 89: 555-563.
23. Engstrom CM, Loeb GE, Reid JG, et al. Morphometry of the human thigh muscles: a comparison between anatomical sections and computer tomographic and magnetic-resonance images. *J Anat.* 1991; 176: 139-156.
24. Franchi MV, Atherton PJ, Reeves ND, et al. Architectural, functional and molecular responses to concentric and eccentric loading in human skeletal muscle. *Acta Physiologica.* 2014; 210: 642-654.
25. Edwards RH, Young A, Hosking GP, et al. Human skeletal muscle function: description of tests and normal values. *Clin Sci Mol Med.* 1977; 52: 283-290.
26. Neder JA, Nery LE, Silva AC, et al. Maximal aerobic power and leg muscle mass and strength related to age in non-athletic males and females. *Eur J Appl Physiol Occup Physiol.* 1999; 79: 522-530.
27. Toft I, Lindal S, Bonna KH, et al. Quantitative measurement of muscle fiber composition in a normal population. *Muscle Nerve.* 2003; 28: 101-108.
28. West DW, Burd NA, Churchward-Venne TA, et al. Sex-based comparisons of myofibrillar protein synthesis after resistance exercise in the fed state. *J Appl Physiol.* 2012; 112: 1805-1813.
29. Fiatarone MA, Marks EC, Ryan ND, et al. High-intensity strength training in nonagenarians. Effects on skeletal muscle. *JAMA.* 1990; 263: 3029-3034.
30. Frontera WR, Hughes VA, Fielding RA, et al. Aging of skeletal muscle: a 12-yr longitudinal study. *J Appl Physiol.* 2000; 88: 1321-1326.
31. Fiatarone MA, O'Neill EF, Ryan ND, et al. Exercise training and nutritional supplementation for physical frailty in very elderly people. *N Engl J Med.* 1994; 330: 1769-1775.
32. Lee JD, Fry CS, Mula J, et al. Aged muscle demonstrates fiber-type adaptations in response to mechanical overload, in the absence of myofibers hypertrophy, independent of satellite cell abundance. *J Gerontol A Biol Sci Med Sci.* 2015; Apr 15 [Epub ahead of print].
33. Bellamy LM, Joannisse S, Grubb A, et al. The acute satellite cell response and skeletal muscle hypertrophy following resistance training. *PLoS One.* 2014; 9:e109739.
34. Farup J, Rahbek SK, Riis S, et al. Influence of exercise contraction mode and protein supplementation on human skeletal muscle satellite cell content and muscle fiber growth. *J Appl Physiol.* 2014; 117: 898-909.
35. Fry CS, Noehren B, Mula J, et al. Fibre type-specific satellite cell response to aerobic training in sedentary adults. *J Physiol.* 2014; 592: 2625-2635.
36. Pallafacchina G, Blaauw B, Schiaffino S. Role of satellite cells in muscle growth and maintenance of muscle mass. *Nutr Metab Cardiovasc Dis.* 2013; 23 Suppl 1: S12-S18.
37. Hanssen KE, Kvamme NH, Nilsen TS, et al. The effect of strength training volume on satellite cells, myogenic regulatory factors, and growth factors. *Scand J Med Sci Sports.* 2013; 23: 728-739.
38. Hawke TJ, Garry DJ. Myogenic satellite cells: physiology to molecular biology. *J Appl Physiol.* 2001; 91: 534-551.
39. Rosenblatt JD, Yong D, Parry DJ. Satellite cell activity is required for hypertrophy of overloaded adult rat muscle. *Mus Nerve.* 1994; 17: 608-613.
40. Vierck J, O'Reilly B, Hossner K, et al. Satellite cell regulation following myotrauma caused by resistance exercise. *Cell Biol Int.* 2000; 24: 263-272.
41. Toigo M, Boutellier U. New fundamental resistance exercise determinants of molecular and cellular muscle adaptations. *Eur J Appl Physiol.* 2006; 97: 642-663.
42. MacDougall JD, Sale DG, Alway SE, et al. Muscle fiber number in biceps brachii in bodybuilders and control subjects. *J Appl Physiol.* 1984; 57: 1399-1403.
43. Zatsiorsky VM. *Science and Practice of Strength Training.* Champaign, IL: Human Kinetics, 1995.
44. Tesch PA, Larsson L. Muscle hypertrophy in bodybuilders. *Eur J Appl Physiol Occup Physiol.* 1982; 49: 301-306.
45. Siff MC, Verkhoshansky YV. *Supertraining.* 4th ed. Denver, CO: Supertraining International, 1999.
46. Wilmore JH. Alterations in strength, body composition and anthropometric measurement consequent to a 10-week weight training-program. *Med Sci Sports Exerc.* 1974; 6: 133-138.
47. Cureton KJ, Collins MA, Hill DW, et al. Muscle hypertrophy in men and women. *Med Sci Sports Exerc.* 1988; 20: 338-344.

48. Welle S, Totterman S, Thornton C. Effect of age on muscle hypertrophy induced by resistance training. *J Gerontol A Biol Sci Med Sci*. 1996; 51: M270-M275.
49. Kadi F, Bonnerud P, Eriksson A, et al. The expression of androgen receptors in human neck and limb muscles: effects of training and self-administration of androgenic-anabolic steroids. *Histochem Cell Biol*. 2000; 113:9.
50. den Hoed MD, Hesselink MKC, Westerterp KR. Skeletal muscle fiber-type and habitual physical activity in daily life. *Scand J Med Sci Sports*. 2009; 19:373-380.
51. Wang YX, Zhang CL, Yu RT, et al. Regulation of muscle fiber type and running endurance by PPARdelta. *PLoS Biol*. 2004; 2:e294.
52. Rodriguez LP, Lopez-Rego J, Calbet JAL, et al. Effects of training status on fibers of the muscles vastus lateralis in professional road cyclists. *Am J Phys Med Rehabil*. 2002; 81:651-660.
53. MacDougall JD, Elder GCB, Sale DG, et al. Effects of strength training and immobilization on human-muscle fibers. *Eur J Appl Physiol Occup Physiol*. 1980; 43: 25-34.
54. Hakkinen K, Komi P, Tesch P. Effect of combined concentric and eccentric strength training and detraining on force-time, muscle fiber and metabolic characteristics of leg extensor muscle. *Scand J Sports Sci*. 1981; 3: 50-58.
55. Lieber RL. *Skeletal Muscle Structure, Function, and Plasticity*. 3rd ed. Philadelphia, PA: Lippincott Williams & Wilkins, 2010.
56. Lieber RL, Friden JO, Hargens AR, Feringa ER. Long-term effects of spinal cord transection on fast and slow rat skeletal muscle. II. Morphometric properties. *Exp Neurol*. 1986; 91: 435-448.
57. Eisenberg BR, Brown JM, Salmons S. Restoration of fast muscle characteristics following cessation of chronic stimulation. The ultrastructure of slow-to-fast transformation. *Cell Tissue Res*. 1984; 238:221-230.
58. Goldspink G. Changes in striated muscle fibres during contraction and growth with particular reference to myofibril splitting. *J Cell Sci*. 1971; 9: 123-128.
59. Patterson S, Goldspink G. Mechanism of myofibril growth and proliferation in fish muscle. *J Cell Sci*. 1976; 22: 607-616.
60. Ashmore CR, Summers PJ. Stretch-induced growth in chicken wing muscles: myofibrillar proliferation. *Am J Physiol*. 1981; 241: C93-C97.
61. Folland JP, Williams AG. The adaptations to strength training: morphological and neurological contributions to increased strength. *Sports Med*. 2007; 37: 145-168.
62. McCall GE, Byrnes WC, Dickinson A, et al. Muscle fiber hypertrophy, hyperplasia, and capillary density in college men after resistance training. *J Appl Physiol*. 1996; 81: 2004-2012.
63. Woo SL, Gomez MA, Amiel D, et al. The effects of exercise on the biomechanical and biochemical properties of swine digital flexor tendons. *Biomech Eng*. 1981; 103:51-56.
64. Kubo K, Kaneshia H, Fukunaga T. Effects of different duration isometric contractions on tendon elasticity in human quadriceps muscles. *J Physiol*. 2001; 536: 649-655.
65. Reeves ND, Maganaris CN, Narici MV. Effect of strength training on human patella tendon mechanical properties of older individuals. *J Physiol*. 2003; 548: 971-981.
66. Kubo K, Kaneshia H, Fukunaga T. Effects of resistance and stretching training programmes on the viscoelastic properties of human tendon structures in vivo. *J Physiol*. 2002; 538: 219-226.
67. Huxley AF. Muscle structure and theories of contraction. *Prog Biophysics Biophysical Chem*. 1957; 7: 255-318.
68. Goldberg AL, Etlinger JD, Goldspink DF, et al. Mechanism of work-induced hypertrophy of skeletal muscle. *Med Sci Sports Exerc*. 1975; 7: 248-261.
69. Kongsgaard M, Aagaard P, Kjaer M, et al. Structural Achilles tendon properties in athletes subjected to different exercise modes and in Achilles tendon rupture patients. *J Appl Physiol*. 2005; 99: 1965-1971.
70. Michna H, Hartmann G. Adaptation of tendon collagen to exercise. *Int Orthop*. 1989; 13: 161-165.
71. Wood TO, Cooke PH, Goodship AE. The effect of exercise and anabolic steroids on the mechanical properties and crimp morphology of the rat tendon. *Am J Sports Med*. 1988; 16: 153-158.
72. Brumitt J. Muscle and Tendon Healing. In Manske RC. *Fundamental Orthopedic Management for the Physical Therapist Assistant*. 4th ed. St. Louis, MO: Mosby/Elsevier, 2015.
73. Lieber RL, Friden JO, Hargens AR, et al. Differential response of the dog quadriceps muscle to external skeletal fixation of the knee. *Muscle Nerve*. 1988; 11; 193-201.
74. Lieber RL, McKee-Woodburb T, Gershuni DH. Recovery of the dog quadriceps after 10 weeks of immobilization followed by 4 weeks of remobilization. *J Orthop Res*. 1989; 7; 408-412.
75. Staron RS. Human skeletal muscle fiber types: delineation, development, and distribution. *Can J Appl Physiol*. 1997;22:307-327.

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76. Pette D, Staron RS. Mammalian skeletal muscle fiber type transitions. *Int Rev Cytol.* 1997; 170:143-223.
 77. Johnson MA, Polgar J, Weightman D, et al. Data on the distribution of fibre types in thirty-six human muscles. An autopsy study. *J Neurol Sci.* 1973; 18: 111-12
 78. Gouzi F, Maury J, Molinari N, et al. Reference values for vastus lateralis fiber size and type in healthy subjects over 40 years old: a systematic review and metaanalysis. *J Appl Physiol.* 2013; 115: 346-354.
 79. Rodríguez LP, López-Rego J, Calbet JA, et al. Effects of training on fibers of the musculus vastus lateralis in professional road cyclists. *Am J Phys Med Rehabil.* 2002; 81:651-660.
 80. Wilson JM, Loenneke JP, Jo E, et al. The effects of endurance, strength, and power training on muscle fiber type shifting. *J Strength Cond Res.* 2012; 26: 1724-1729.
 81. Trappe S, Harber M, Creer A, Gallagher P, Slivka D, Minchev K, Whitsett D. Single muscle fiber adaptations with marathon training. *J Appl Physiol.* 2006; 101: 721-727.
 82. Lovering RM, Russ DW. Fiber type composition of cadaveric human rotator cuff muscles. *J Orthop Sports Phys Ther.* 2008; 38: 674-680.
 83. Engel WK. Fiber-type nomenclature of human skeletal muscle for histochemical purposes. *Neurology.* 1974; 24: 344-348.
 84. Ng JK, Richardson CA, Kippers V, Parnianpour M. Relationship between muscle fiber composition and functional capacity of back muscles in healthy subjects and patients with back pain. *J Orthop Sports Phys Ther.* 1998; 27: 389-402.
 85. Fukashiro S, Komi PV, Jarvinen M, et al. In vivo Achilles tendon loading during jumping in humans. *Eur J Appl Physiol Occup Physiol.* 1995; 71: 453-458.
 86. Platt MA. Tendon repair and healing. *Clin Podiatr Med Surg.* 2005; 22: 553-560.
 87. Kongsgaard M, Reitelseder S, Pedersen TG, Holm L, Aagaard P, Kjaer M, Magnusson SP. *Acta Physiol.* 2007; 191: 111-121.
 88. Reeves ND, Maganaris CN, Narici MV. Effect of strength training on human patella tendon mechanical properties of older individuals. *J Physiol.* 2003; 548: 971-981.
 89. Leach RE, James S, Wasilewski S. Achilles tendinitis. *Am J Sports Med.* 1981; 9: 93-98.
 90. Alfredson H, Lorentzon R. Chronic Achilles tendinosis: recommendations for treatment and prevention. *Am J Sports Med.* 2000; 29: 135-146.
 91. Pasquina PF, Griffin SC, Anderson-Barnes VC, et al. Analysis of injuries from the Army Ten Miler: a 6-year retrospective review. *Mil Med.* 2013; 178: 55-60.
 92. Daoud AI, Geissler GJ, Wang F, et al. Foot strike and injury rates in endurance runners: a retrospective study. *Med Sci Sports Exerc.* 2012; 44: 1325-1334.
 93. Bennell KL, Malcolm SA, Thomas SA, et al. The incidence and distribution of stress fractures in competitive track and field athletes. A twelve-month prospective study. *Am J Sports Med.* 1996; 24: 211-217.
 94. Cook JL, Khan KM, Purdam C. Achilles tendinopathy. *Man Ther.* 2002; 7: 121-130.
 95. Paavola M, Kannus P, Paakkala T, Pasanen M, Jarvinen M. Long-term prognosis of patients with Achilles tendinopathy. An observational 8-year follow-up study. *Am J Sports Med.* 2000; 28: 634-642.
 96. Sayana MK, Maffulli N. Eccentric calf muscle training in non-athletic patients with Achilles tendinopathy. *J Sci Med Sport.* 2007; 10: 52-58.
 97. Alfredson H. Chronic midportion Achilles tendinopathy: an update on research and treatment. *Clin Sports Med.* 2003; 22: 727-741.
 98. Rompe JD, Furia J, Maffuli N. Eccentric load versus eccentric loading plus shock-wave treatment for midportion Achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* 2009; 37: 463-470.
 99. van der Plas A, de Jonge S, de Vos RJ, et al. A 5-year follow-up study of Alfredson's heel-drop exercise programme in chronic midportion Achilles tendinopathy. *Br J Sports Med.* 2012; 46: 214-218.
 100. Abbassian A, Khan R. Achilles tendinopathy: pathology and management strategies. *Br J Hosp Med.* 2009; 70: 519-523.
 101. Beyer R, Kongsgaard M, Hougs KB, et al. Heavy slow resistance versus eccentric training as treatment for Achilles tendinopathy: a randomized controlled trial. *Am J Sports Med.* 2015; 43: 1704-1711.