

How Tendons Buffer Energy Dissipation by Muscle

Thomas J. Roberts and Nicolai Konow

AQ1 Department of Ecology and Evolutionary Biology, Brown University, Providence, RI

ROBERTS, T.J. and N. KONOW. How tendons buffer energy dissipation by muscle. *Exerc. Sport Sci. Rev.*, Vol. 41, No. 4, pp. 00–00, 2013. *To decelerate the body and limbs, muscles lengthen actively to dissipate energy. During rapid energy-dissipating events, tendons buffer the work done on muscle by storing elastic energy temporarily, then releasing this energy to do work on the muscle. This elastic mechanism may reduce the risk of muscle damage by reducing peak forces and lengthening rates of active muscle*

AQ2 **Key Words:** muscle, tendon, elastic energy, energy dissipation, deceleration

INTRODUCTION

Tendons play a critical role in enhancing muscle performance for many activities. In running, their springlike function can reduce the work muscles must do to maintain the cyclic motion of the body and limbs (21). For high-power activities like jumping or acceleration, the rapid release of energy stored in tendon can provide power outputs that exceed the power-producing capacity of muscles (21). The goal of this review is to address the role of tendons in an activity that is critical for locomotion but relatively poorly understood: energy dissipation. Muscles dissipate energy when they lengthen actively, and energy dissipation is required for any activity involving the deceleration of the body or limbs, including quick maneuvers, reducing speed in walking or running, or in landing from a jump. The ability of muscles to dissipate energy undoubtedly affects a wide range of locomotor activities, and it is during these dissipative activities that injuries tend to occur. Fractured bones, torn ligaments, and pulled muscles are often associated with rapid maneuvers that involve deceleration and dissipation of mechanical energy. The risk of injury likely depends on the mechanical performance of muscles during rapid decelerations, thus, it is worth understanding how muscles perform these tasks and how the springlike action of tendons may influence muscle mechanical behavior.

Among the studies that have addressed the role of tendons in energy-dissipating activities, there generally is a consensus that tendons act as a mechanical buffer to protect muscles from possible damage associated with rapid active stretching (8,10,19,20). Here, we present the central hypothesis that tendons protect muscle from the damage associated with eccentric contractions by delaying and reducing energy absorption by active muscle fibers. We propose that, although the temporary storage of energy in tendons does not reduce muscle lengthening significantly, it reduces the chance of damage by allowing for muscle contractions that are slower, less powerful, and involve lower forces.

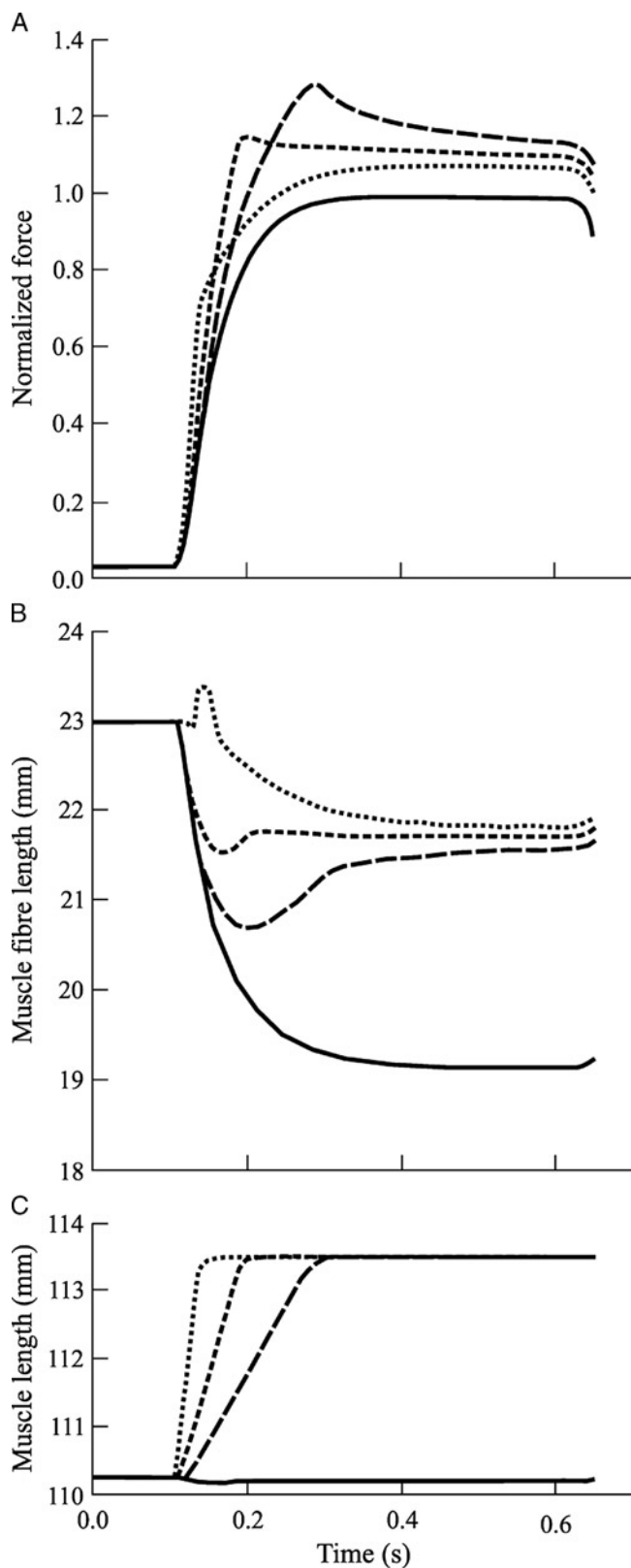
TENDONS AS BUFFERS: ESTABLISHED IDEAS AND LINGERING QUESTIONS

The idea of tendons as a mechanical buffer was proposed by Griffiths (8), based on observations of the mechanical behavior of isolated cat muscles. In an elegant series of experiments, Griffiths (8) took advantage of a then-new technology, sonomicrometry, to make measurements of the length changes in muscle fascicles independently of the length change applied to the whole muscle-tendon unit (MTU) by an external servomotor (8). The key observation was a relatively simple but striking one: when a ramp lengthening was applied to the MTU simultaneous with the onset of muscle activation, there was little, if any, lengthening of the muscle fascicles (Fig. 1). In fact, during all but the fastest lengthening trials the muscle fascicles shortened exclusively while the MTU lengthened. The observation that an MTU lengthening could be accommodated by tendon elasticity was consistent with observations of the behavior of ankle extensors during the stance phase of walking and hopping (7,9), but measurements by Griffiths (8) on isolated muscles showed just

Address for correspondence: Thomas J. Roberts, Ph.D., Department of Ecology and Evolutionary Biology, Brown University, Providence RI 02912 (E-mail: Thomas_Roberts@Brown.edu).

Accepted for publication: May 15, 2013.
Associate Editor: Daniel P. Ferris, Ph.D.

0091-6331/4104/00–00
Exercise and Sport Sciences Reviews
Copyright © 2013 by the American College of Sports Medicine



AQ3 **Figure 1.** Muscle force (A), fiber (fascicle) length (B), and whole muscle-tendon unit (MTU) length (C) for cat medial gastrocnemius stimulated to contract *in situ*. During MTU lengthening, muscle fascicles shortened (*solid and dashed lines*) or were lengthened at a much slower rate compared with the whole muscle (*dotted line, fastest MTU lengthening*). (Reprinted from (8). Copyright © 1991 Wiley. Used with permission.)

how substantial this effect could be. The study suggested that the reduction in muscle lengthening caused by tendon elasticity might serve a protective effect, given muscle's propensity to damage during lengthening, and concluded that the tendon "acts as a mechanical buffer" (8).

Relatively recent advances in ultrasound techniques for measuring muscle fascicle and tendon displacements also have provided evidence of a tendon-buffering mechanism. Reeves and Narici (19) measured muscle fascicle length changes as subjects performed voluntary contractions during isokinetic joint excursions controlled by a dynamometer. Across a range of lengthening velocities, there was negligible change in the length of the fascicles measured from rest to a given point in joint angular excursion. This observation led the authors to conclude that stretch of the tendon during the period of MTU lengthening allowed the muscle fascicles to behave nearly isometrically. The study concluded that tendon acts as a mechanical buffer during eccentric (lengthening) contractions, possibly as a protective mechanism against muscle damage.

The idea that tendons act like springy buffers to reduce impact is appealing and intuitive, and it evokes easy analogies to the cushioning effect of a car's springy suspension or a gymnast's landing mat. But the details of both the structural arrangement of muscles and tendons and their mechanical behavior raise several questions about how a tendon buffering mechanism might work. For example, how does a structure that cannot dissipate energy act to cushion an energy-dissipating event? Tendons are quite resilient springs — they return 90% to 97% of any energy they absorb — so, although they can store energy temporarily, this stored energy must be returned. During a movement like walking or running, energy stored in tendon can be used subsequently to drive joint motion and work, but in an activity requiring energy dissipation, the recoil must drive active muscle lengthening. In the examples given above, muscle fascicle lengthening was not observed during the stretch of an MTU but, undoubtedly, it occurred during the period of the contraction when force declined and energy was released from stretched tendon. Thus, several important questions remain if we are to understand how tendons might provide a protective effect for muscle. Our own studies of muscle-tendon function during energy-dissipating activities have been motivated to answer some of these questions. If tendon only delays the eccentric activity of muscle fascicles, how can this reduce the chance of injury? Does the near-isometric or shortening behavior of muscle fascicles observed for lengthening contractions during controlled driven contractions occur during ordinary movements? What are the implications of this mechanism for neural control?

TENDONS DELAY ENERGY ABSORPTION BY MUSCLES

In Situ Measurements of Muscle Lengthening

We have examined the role of tendons in energy-dissipating events using wild turkeys as a model. Although arguably not the most obvious study animal for locomotor research, turkeys offer a unique advantage: segments of their limb tendons ossify, and these stiff tendon regions allow the

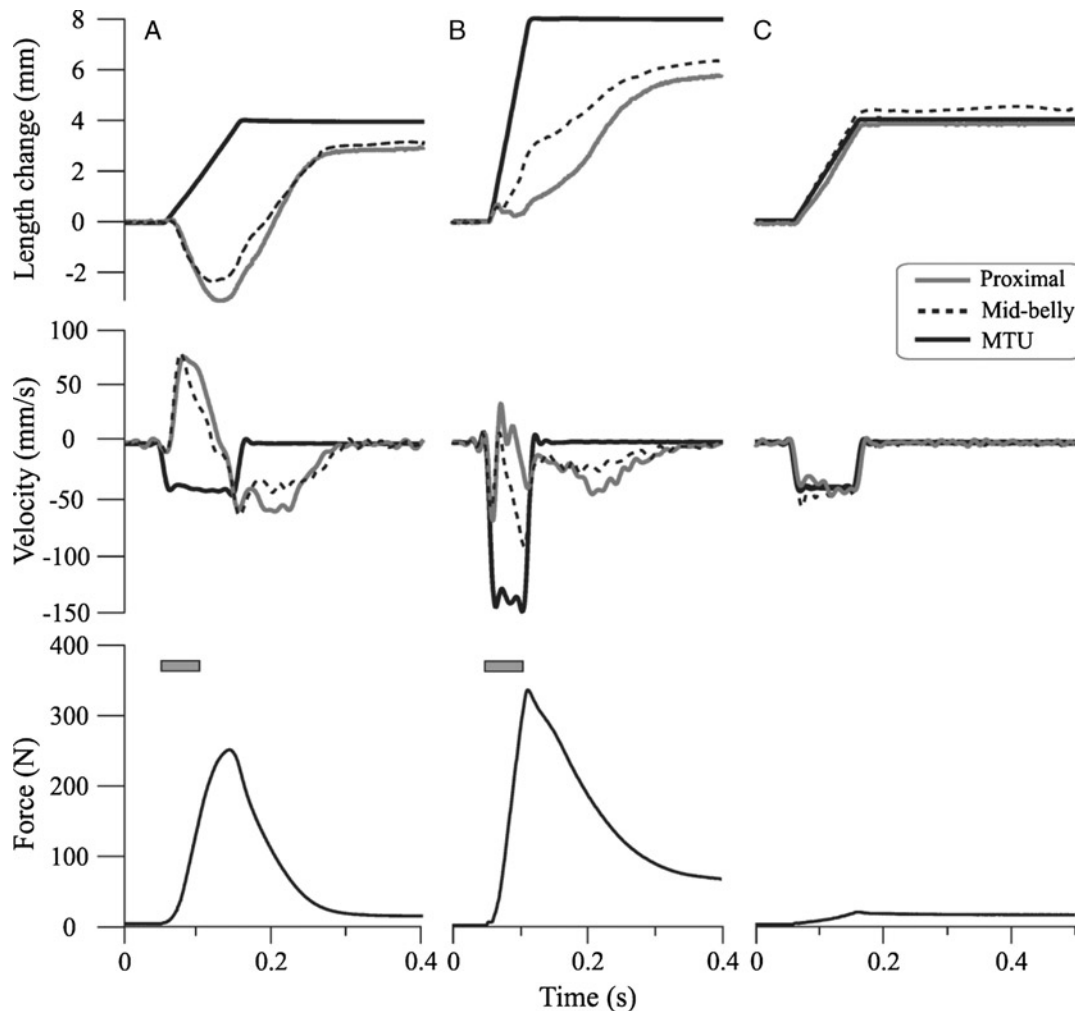
application of foil element strain gauges that can be used to measure the force produced by individual muscles during locomotor activities (strain measurements are converted easily to force after an *in situ* calibration of the tendon-mounted strain gauges (6)). The lateral gastrocnemius muscle in turkeys has the same action as the gastrocnemius in humans, ankle extension (*i.e.*, plantarflexion), and it is loaded eccentrically when the ankle is flexed by an external load. In turkeys, the muscle can be studied conveniently both *in vivo*, during locomotor activities, and *in situ*, where muscle stimulation and length can be controlled.

We studied the mechanical behavior of the turkey gastrocnemius during active lengthening *in situ* using a servomotor to measure MTU length and force and sonomicrometry to measure muscle fascicle length (20). In a series of contractions, a constant velocity stretch was applied to the muscle, with the onset of stretch occurring simultaneously with the onset of muscle stimulation (Fig. 2). Results from these contractions showed that both the MTU and muscle fascicles underwent net lengthening, but the timing of the two was completely out of phase. During the period of MTU lengthening, muscle

fascicles underwent minimal lengthening or, in most cases, shortened. Most or all of the fascicle lengthening occurred later, after the MTU lengthening ramp, during a period when the MTU was isometric. This pattern reflects the predominant effect of tendon stretch and recoil. Lengthening of the MTU is accommodated initially by tendon stretch (and, for slow lengthening contractions, tendon stretch also requires fascicle shortening), which occurs during the rapid rise in force associated with muscle activation. Later, when the MTU is isometric, relaxation of the muscle allows for a decay in muscle force and recoil of the elastic tendon. This tendon shortening drives muscle fascicle lengthening, although the entire MTU does not change length during this period. Thus, there is an internal shuttling of energy where strain energy first loaded into stretching tendon is later released to lengthen active muscle fibers, where it is dissipated (20).

In Vivo Measurements of Muscle Lengthening

In situ experiments provide insight because they describe the mechanical behavior of maximally active muscles driven



AQ3 Figure 2. Length and force in turkey gastrocnemius muscle fascicles and whole muscle-tendon unit (MTU) during moderate (A), rapid (B), and passive (C) lengthening. Muscle fascicles shorten during moderate lengthening. In active contractions, most of the muscle lengthening occurs when the MTU is isometric, indicating that it is driven by tendon recoil. During passive lengthening, forces are low and muscle fascicle length changes correspond to length changes of the whole MTU. (Reprinted from (19). Copyright © 2003 American Physiological Society. Used with permission.)

by an external load, but this behavior is not necessarily representative of the events that occur in ordinary movements. We used the same muscle studied *in situ*, the turkey lateral gastrocnemius, to study how MTU absorb energy during an activity that requires substantial energy dissipation: landing from a jump (10). To get repeatable behavior, we used a rope and pulley system to suspend animals over a landing area, then released them at a height that allowed for controlled coordinated landings. Results from this study confirmed that tendons play a role in buffering the initial rapid absorption of energy. During the first period after foot contact, when muscle force rose rapidly, energy was stored almost exclusively in the lateral gastrocnemius tendon. It was only later, after most of the ankle flexion had occurred, that the muscle fascicles lengthened. Because tendon energy was stored rapidly but released slowly, the peak rate of energy absorption into the tendon was much greater than the peak rate of energy absorption by the muscle (10).

Studies of muscle function in humans also have provided evidence of the importance of tendon elasticity during activities involving eccentric muscle contraction. Spanjaard and colleagues (23) used ultrasound to track muscle fascicle length in the medial gastrocnemius (MG) of human subjects walking up and down stairs. During stair descent, the pattern of muscle fascicle length change was consistent with observations from isolated animal muscles. Muscle fascicle and MTU length changes were out of phase, with most of the fascicle lengthening occurring late in the step when the MTU underwent little change in length, providing evidence for the significant influence of tendon. In a study of a more complex motion, a drop-jump on a sled, Sousa and coworkers (22) also found that the MG initially shortened while the MTU lengthened at impact. They found that the soleus, however, lengthened early in the event during MTU lengthening. It should be noted that this was a somewhat more complex motor behavior and not purely energy dissipative, as subjects performed a stretch-shorten cycle to propel themselves upward along the sled after the initial impact (22).

PROTECTIVE ROLE OF TENDON FUNCTION

To dissipate energy, muscle fascicles must lengthen actively. Both *in situ* and *in vivo* studies indicate that tendons can delay this lengthening during energy-dissipating events by absorbing impact energy transiently and then releasing energy to do work on muscle fascicles. Because tendons are so resilient, this mechanism does not alter the amount of muscle fascicle lengthening required to dissipate energy significantly, only its timing. Can altering the timing of muscle energy dissipation provide a protective effect? Our studies of turkey gastrocnemius function *in situ* and during ordinary movements point to several possible mechanisms whereby tendon elasticity may protect muscle and other structures from damage.

Perhaps the most important effect of tendon elasticity is that it reduces the force developed in an eccentric contraction. If a muscle is stimulated maximally, as in an *in situ* preparation, the rate of increase in force after the onset of stimulation is determined largely by force-velocity effects and the speed of

shortening of active fibers (5). Muscle shortening will reduce the rate of rise in force, whereas muscle lengthening will increase it. This effect is apparent in the contractions shown in Figure 2, where force rises rapidly in the fast stretch (Fig. 2B) compared with the slower stretch (Fig. 2A). For a fixed duration of stimulation, these differences in force rise rate result in differences in the peak force developed. In turkey gastrocnemius *in situ*, we found that the peak force of contraction was only about half of what it would have been had the stretch been applied directly to the muscle fascicles rather than to the tendon (19). *In vivo*, the lack of fascicle lengthening during the prominent early burst of electromyographic activity (Fig. 3) presumably had a similar effect of limiting the peak muscle force developed, relative to the force that would have been developed had the muscle fascicles lengthened during this period. This mechanism would seem to be particularly important for landings, where maximal muscle recruitment, for example, to deal with an impact of uncertain intensity, might lead to

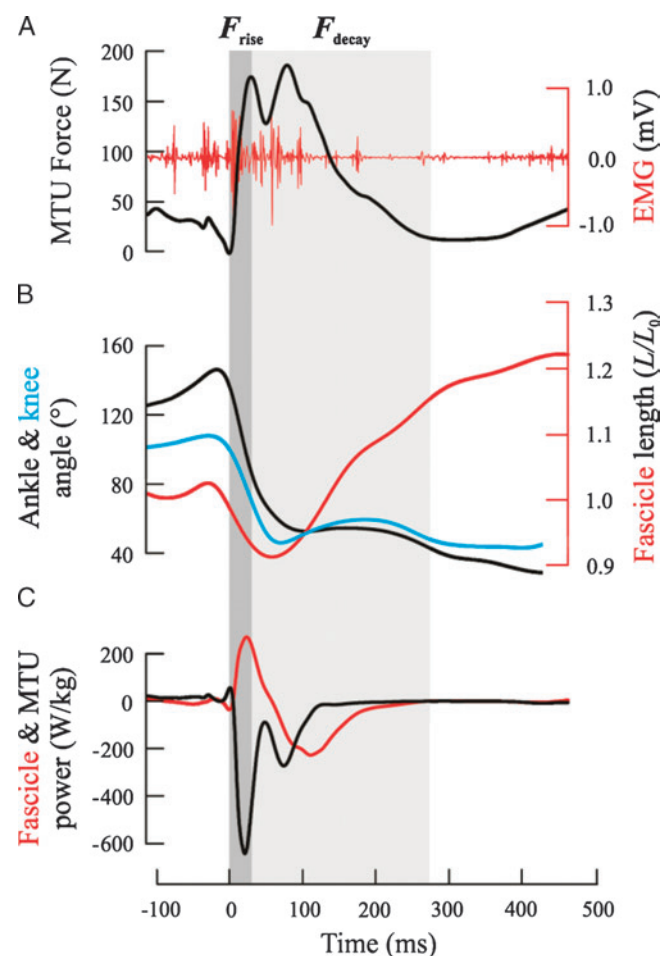


Figure 3. Mechanical behavior of the turkey lateral gastrocnemius during a drop landing. During the early part of the contraction, muscle fascicles shorten (B) at the same time the muscle-tendon unit (MTU) lengthens because of ankle flexion. Power (C) flows initially into the MTU and later into the muscle fascicles after most of the joint movement is complete. The delay in energy transfer to the muscle fascicles, caused by transient tendon energy storage, results in attenuation of the power absorption by muscle fascicles. (Reproduced from (10). Copyright © 2011 American Physiological Society. Used with permission.)

very high forces if muscle fascicles also were lengthened rapidly during activation.

Two other possible protective roles of tendons resulted from the delay in energy absorption by muscle fascicles during MTU lengthening. We found that both *in vivo* and *in situ*, the tendon stretched more rapidly than it recoiled. As a result, the rate of lengthening of the muscle fascicles was reduced relative to what it would have been had the initial stretch of the MTU been accommodated by muscle fascicles rather than tendon. The lower rate of fascicle lengthening relative to that of the tendon also tended to reduce the rate of energy absorption, measured as muscle power, in active muscle fascicles. The peak rate of energy absorption by the whole MTU was much greater than that of the muscle because of the very rapid absorption of energy by the tendon in the early part of the contraction. Because this energy was then released relatively slowly to lengthen active muscle fascicles, the rate of energy dissipation by muscle fascicles was lower than the initial rate of energy storage by tendon. This result suggests that, without an elastic tendon, power inputs to active muscle fibers would be much higher (apparent when comparing MTU power during force rise with fascicle power during force decay in Fig. 3C).

Does a reduction in peak force, rate of lengthening, or peak power input to active muscle fibers reduce the chance of muscle damage? A challenge in answering this question is a lack of clear consensus on the factors that determine the risk of muscle damage in an eccentric contraction (18). It seems reasonable to assume that reductions in peak force would reduce the chance of damage not only to muscle but also to all musculoskeletal structures, including bone, ligaments, and other joint connective tissues. Some isolated muscle studies support a link between peak muscle force and the degree of damage from eccentric contractions (12,25), but others suggest that it is maximum fascicle strain, not force, that determines muscle damage during lengthening (11). Some studies have found that increasing the rate of lengthening for an eccentric contraction of fixed strain magnitude increases damage to isolated muscles (25). If so, a tendon mechanism that leads to a reduction in muscle lengthening rate may reduce the chance of muscle damage.

TENDON BUFFERING OF ECCENTRIC CONTRACTIONS: A SUMMARY OF THE MECHANISM

Evidence from both *in situ* and *in vivo* studies suggests that the storage and release of elastic strain energy in tendon can delay and slow the dissipation of mechanical energy by active muscle fascicles. Figure 4 summarizes how this mechanism works at the level of the MTU, and here we describe how it is facilitated by the contractile behavior of skeletal muscle.

F4 Energy absorption is initiated when an external load is applied to the MTU. In landing from a jump, the load would result from the impact force and the joint flexion that accompanies it. The muscle is activated and, as force rises, tendon is stretched and stores energy. In the idealized model in Figure 4, MTU lengthening is accommodated entirely by the tendon stretch, whereas the muscle remains isometric. This event occurs rapidly, and the effective shuttling of energy to the tendon, rather than to

stretching muscle fibers, relies on two physiological properties of the muscle: 1) rapid activation and 2) force-velocity behavior. It is the rapid rise of force that results in a rapid stretch of the tendon, and this occurs because the onset of activation of the muscle is close to simultaneous with the onset of MTU lengthening. A stretch applied to an MTU that is already fully active, in tetanus, results in less tendon lengthening and more muscle fascicle lengthening (8). The force-velocity property of muscle influences the relative contribution of muscle fascicle versus tendon to the MTU lengthening event. Any tendency to lengthen muscle fascicles will lead to an increase in force developed by those fascicles, and increases in force increase the stretch of tendon. This behavior can explain how tendon stretch accommodates MTU stretch across a wide range of velocities: faster stretches tend to lead to higher forces (because of force-velocity effects), and higher forces result in more tendon stretch. Increases in the rate of MTU lengthening tend to reduce the rate of muscle fascicle shortening, but the influence is relatively weak; the slope of the regression of fascicle lengthening/shortening rate versus MTU lengthening rate was less than 0.5 across a wide range of lengthening speeds *in situ* (20). This behavior may make the system very robust in its ability to protect muscle fascicles from lengthening for a wide range of unpredictable impact conditions.

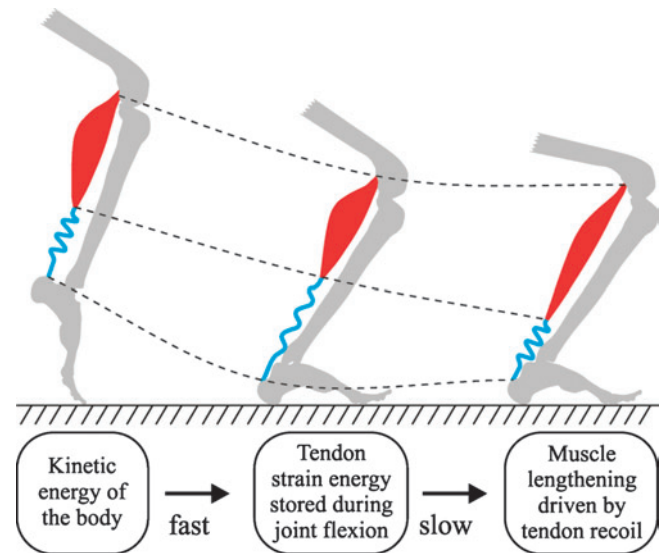


Figure 4. Schematic summary of the tendon buffering mechanism. Energy is absorbed initially by the tendon during a brief and rapid event, followed by a relatively slow flow of energy from the tendon to the muscle as fascicle lengthens and dissipates energy.

Fig 4 4/C

AQ3

is reduced relative to the power input to the tendon that occurs during initial energy storage. Thus, this mechanism works like the familiar power amplifier action of tendon during ballistic activities like jumping, but in reverse.

Muscles with large tendons usually are pennate, meaning the fascicles run at an angle to the line of action of the muscle. During contraction, length changes in pennate muscles result not only from fascicle shortening or lengthening but also from the rotation of fascicles that occurs with changes in pennation angle (3,13,15). During shortening, changes in pennation angle can increase muscle shortening relative to fascicle shortening (3). Changes in pennation angle likely also act during lengthening to increase MTU lengthening relative to fascicle lengthening. It is likely that changes in pennation angle also play a role in protecting muscle fibers from potentially damaging lengthening and may contribute to the length change patterns that have been observed in the early part of contractions for energy-dissipating activities.

BUFFERS, ATTENUATORS, AND SHOCK ABSORBERS — WHAT CAN TERMINOLOGY TEACH US?

Several different terms have been applied to describe the mechanical role of tendons during muscle energy absorption, including a “mechanical buffer,” “shock absorber,” and “power attenuator” (8,10,19,20). Exploring the extent to which physical analogs are or are not accurate can be instructive and can serve our understanding of how the mechanism works.

Both the terms “buffer” and “shock absorber” accurately describe the observation that tendon stretch initially absorbs the energy of impact, thereby reducing, at least for this initial period, the energy that must be absorbed by muscle. Engineered mechanical buffers and shock absorbers, such as the shock absorbers on automobiles, also tend to blunt the force of impact because they slow decelerations. Likewise, our results for turkey gastrocnemius indicate that force of contraction is reduced because of the influence of tendon elasticity on muscle speed of shortening or lengthening. However, the analogy of a buffer or shock absorber is somewhat problematic because engineered devices not only absorb impact energy, they also dissipate it via viscous damping mechanisms. Tendons dissipate only a small fraction (<10%) of the energy they absorb, and the “shock” that they absorb at impact must be released to the muscle eventually, which dissipates the energy.

In the turkey gastrocnemius, the temporary storage and release of energy from tendon to muscle can result in a reduction in the rate at which energy is dissipated by the muscle fascicles. We have referred to this role of tendon as that of a power attenuator because the peak rate of power input to the muscle is reduced. This terminology is appealing because it serves as an intuitive counterpart to the more familiar analogy of tendon as a “power amplifier,” where the storage and release of muscle work by tendon allow for power outputs that exceed the maximum power output of the muscle (e.g., Aerts (1) and Vogel (24)). Both terms have been criticized reasonably because familiar electronic amplifiers work by adding energy to a signal. Tendons neither add nor subtract significant energy; they can

act to increase power output or decrease the rate of power input to muscle only by redistributing work in time. It has been suggested that a more appropriate physical analog for the action of tendons during an energy-dissipating event is a capacitor. Capacitors temporarily store electrical energy and can operate with different time constants for storage and release of this energy. They are used, for example, in electrical signal conditioning to smooth out rapid fluctuations in power. The analogy of a capacitor for the mechanical function of tendons during muscle energy dissipation is appealing because, like a tendon, a capacitor alters power by changing the timing of energy flow, not by adding or subtracting energy. Thus, the idea that tendons act as biological capacitors may provide a more accurate analogy than the notion of amplifiers and attenuators. In our experience, challenges of this analogy are that, in the digital age, a capacitor’s action is too distant from many biologists’ intuition and the necessity of explaining how a capacitor works hinders its utility as a descriptive analogy.

AQ5

IMPLICATIONS FOR NEURAL CONTROL

It has been demonstrated in three different systems that a rapid stretch applied to a fully active muscle initially can be taken up almost entirely by stretching tendon (8,19,20). Increasing the rate of MTU lengthening only increases the force and, therefore, the stretch of the tendon, thus, in practice, it is almost not possible to get a fully active muscle to lengthen in the early part of a contraction. Data from drop landing experiments tend to support this observation because fascicles in turkey lateral gastrocnemius undergo little or no lengthening early in the contraction. This does not mean, however, that muscle fascicles always undergo this kind of contraction pattern. During downhill running, the turkey gastrocnemius undergoes nearly constant velocity lengthening, beginning at toe down and the onset of active force (e.g., Fig. 6 in Gabaldón *et al.* (6)). The different lengthening patterns during running versus drop landings suggest that control of the level of muscle activation can allow modulation of the extent to which the tendon acts as a mechanical buffer. The recruitment of fewer motor units means lower forces and less tendency to stretch the tendon. Griffiths (8) demonstrated the role of muscle recruitment on tendon action in cat gastrocnemius by comparing the fascicle length pattern when an animal scratched its ear to the pattern during a paw shake, elicited by placing tape on the animal’s foot. In paw shake, a relatively high-force activity, length changes in the MTU were out of phase with fascicle length changes, whereas in the low-force ear scratch, there was good agreement at all times between length change in the fascicles and the whole MTU (8).

AQ6

The behavior of the turkey lateral gastrocnemius during a drop landing and *in situ* suggests that the interaction between muscle and tendon mechanical behavior may allow for modulation of the energy-absorbing event by mechanical, rather than neural, feedback. As demonstrated *in situ*, as an increasing rate of stretch of the MTU tends to stretch muscle fascicles, muscle force increases because of force-velocity effects and tendon is stretched more. For activities where the magnitude of

impact force is uncertain or when maximal energy dissipation is desired (such as in a maximal braking step to decelerate in a run), muscles may be recruited maximally. Muscle contractile behavior would then depend on the mechanical events after impact. If the impact energy is low, the muscle likely would shorten against tendon stretch, limiting muscle forces caused by force-velocity effects. Higher impact energies would reduce muscle shortening, resulting in significant tendon stretch, high forces, and greater energy dissipation.

The integrated mechanical behavior of muscle and tendon during high-power eccentric activities also may provide an explanation for puzzling results from studies of eccentric contraction in human subjects. In dynamometer studies, the maximal force developed when active MTU are loaded eccentrically by joint flexion is similar to or only slightly greater than that of an isometric contraction (reviewed in Duchateau and Enoka (4)). Isolated supramaximally active muscle fibers develop forces during lengthening that are 70% to 100% greater than the force developed isometrically, so why don't eccentric contractions in dynamometer studies elicit higher forces? It has been proposed that these low forces result from a neural mechanism that acts to limit force via inhibitory feedback that reduces muscle recruitment during maximal lengthening events. A slight reduction, by about 10%, in electromyographic signal during eccentric loading lends some support to this hypothesis (2). Data from our *in situ* studies suggest an alternative explanation: eccentric loading during motor vehicle crashes stretches the tendon and not the muscle fascicles, and because the fibers are close to isometric, the muscle produces no more force than it would in a contraction in which the joint does not move. The isometric behavior of human tibialis anterior fascicles during lengthening is consistent with this explanation (19). This mechanical force-limiting mechanism also is apparent during *in situ* lengthening of maximally activated turkey gastrocnemius. Given typical force-velocity curves for lengthening muscle (17) and the maximal shortening velocity for turkey lateral gastrocnemius (16), this muscle would be expected to produce a force of about $1.8\times$ the peak isometric tetanic force (P_{0}) when lengthened at a rate of 1 L s^{-1} or faster. When the lateral gastrocnemius MTU was lengthened at 1.6 L s^{-1} , peak force was approximately equal to P_{0} , the maximal force in an isometric contraction. The very fastest lengthening rate, 4.5 L s^{-1} , produced a force that exceeded P_{0} , but only by about 30%, not the 80% difference expected for an isotonic lengthening contraction. These results suggest that the mechanical influence of tendon serves to limit muscle forces significantly, and this mechanism may provide a mechanical, rather than a neural, basis for the force-limiting mechanism in maximal eccentric contractions in human subjects.

An intriguing aspect of the central role of tendons in energy-dissipating activities is that it provides a potential mechanism for the very rapid adaptation to eccentric activities that has been observed in human subjects. A single bout of eccentric exercise can protect against damage in subsequent eccentric bouts (13). The mechanism for this "repeated bout" effect is unclear, and the rapidity of the response challenges explanations that require significant remodeling of cells and tissues. We hypothesize that a learned ability to fine-tune muscle activation patterns so as to enhance the protective effect of tendon elasticity may occur rapidly and may

explain the significant reduction in eccentric muscle damage after an initial bout of experience with a given eccentric activity. This possible mechanism of neural adaptation to eccentric exercise has not yet been tested.

CONCLUSIONS

Under circumstances that require the dissipation of mechanical energy, the elastic behavior of tendon can protect muscle by storing energy rapidly temporarily, then releasing it more slowly to do work on the muscle. This mechanism may protect muscle from damage by reducing the rate of muscle fascicle lengthening and peak power inputs to muscle fascicles.

Acknowledgments

This work was supported by the National Institutes of Health AR and National Science Foundation.

Insightful comments and suggestions from three anonymous reviewers significantly improved the article. The authors gratefully acknowledge support from the National Science Foundation (IOS0642428) and the National Institute of Arthritis and Musculoskeletal and Skin Diseases of the National Institutes of Health under Award AR055295. The content is solely the responsibility of the authors and necessarily does not represent the official views of the National Institutes of Health.

References

1. Aerts P. Vertical jumping in *Galago senegalensis*: the quest for an obligate mechanical power amplifier. *Philos. Trans. Royal Soc. B.* 1997; 353: 1607–20.
2. Beltman JG, Sargeant AJ, van Mechelen W, de Haan A. Voluntary activation level and muscle fiber recruitment of human quadriceps during lengthening contractions. *J. Appl. Physiol.* 2004; 97(2):619–26.
3. Brainerd EL, Azizi E. Muscle fiber angle, segment bulging and architectural gear ratio in segmented musculature. *J. Exp. Biol.* 2005; 208(Pt 17): 3249–61.
4. Duchateau J, Enoka RM. Neural control of shortening and lengthening contractions: influence of task constraints. *J. Physiol.* 2008; 586(Pt 24): 5853–64.
5. Edman KA, Josephson RK. Determinants of force rise time during isometric contraction of frog muscle fibres. *J. Physiol.* 2007; 580(Pt. 3): 1007–19.
6. Gabaldón AM, Nelson FE, Roberts TJ. Mechanical function of two ankle extensors in wild turkeys: shifts from energy production to energy absorption during incline versus decline running. *J. Exp. Biol.* 2004; 207(Pt 13):2277–88.
7. Griffiths RI. The mechanics of the medial gastrocnemius muscle in the freely hopping wallaby (*Thylogale billiardieri*). *J. Exp. Biol.* 1989; 147:439–56.
8. Griffiths RI. Shortening of muscle fibers during stretch of the active cat medial gastrocnemius muscle: the role of tendon compliance. *J. Physiol. Lond.* 1991; 436:219–36.
9. Hof AL, Geelen BA, Van den Berg J. Calf muscle moment, work and efficiency in level walking; role of series elasticity. *J. Biomech.* 1983; 16(7):523–37.
10. Konow N, Azizi E, Roberts TJ. Muscle power attenuation by tendon during energy dissipation. *Proc. Biol. Sci.* 2011; 279(1731):1108–13.
11. Lieber RL, Fridén J. Muscle damage is not a function of muscle force but active muscle strain. *J. Appl. Physiol.* 1993; 74(2):520–6.
12. McCully KK, Faulkner JA. Characteristics of lengthening contractions associated with injury to skeletal muscle fibers. *J. Appl. Physiol.* 1986; 61(1):293–9.
13. McHugh MP. Recent advances in the understanding of the repeated bout effect: the protective effect against muscle damage from a single bout of eccentric exercise. *Scand. J. Med. Sci. Sports.* 2003; 13(2):88–97.

14. Muh ZF. Active length-tension relation and the effect of muscle pinnation on fiber lengthening. *J. Morphol.* 1982; 173(3):285–92.
15. Narici MV, Binzoni T, Hiltbrand E, Fasel J, Terrier F, Cerretelli P. *In vivo* human gastrocnemius architecture with changing joint angle at rest and during graded isometric contraction. *J. Physiol.* 1996; 496(1):287–97.
16. Nelson FE, Gabaldon AM, Roberts TJ. Force-velocity properties of two avian hindlimb muscles. *Comp. Biochem. Physiol. A. Mol. Integr. Physiol.* 2004; 137(4):711–21.
17. Otten E. A myocybernetic model of the jaw system of the rat. *J. Neurosci. Meth.* 1987; 21:287–302.
18. Proske U, Morgan DL. Muscle damage from eccentric exercise: mechanism, mechanical signs, adaptation and clinical applications. *J. Physiol.* 2001; 537(Pt 2):333–45.
19. Reeves ND, Narici MV. Behavior of human muscle fascicles during shortening and lengthening contractions *in vivo*. *J. Appl. Physiol.* 2003; 95(3):1090–6.
20. Roberts TJ, Azizi E. The series-elastic shock absorber: tendons attenuate muscle power during eccentric actions. *J. Appl. Physiol.* 2010; 109(2): 396–404.
21. Roberts TJ, Azizi E. Flexible mechanisms: the diverse roles of biological springs in vertebrate movement. *J. Exp. Biol.* 2011; 214(Pt 3):353–61.
22. Sousa F, Ishikawa M, Vilas-Boas JP, Komi PV. Intensity- and muscle-specific fascicle behavior during human drop jumps. *J. Appl. Physiol.* 2007; 102(1):382–9.
23. Spanjaard M, Reeves ND, van Dieën JH, Baltzopoulos V, Maganaris CN. Gastrocnemius muscle fascicle behavior during stair negotiation in humans. *J. Appl. Physiol.* 2007; 102(4):1618–23.
24. Vogel S. Living in a physical world III. Getting up to speed. *J. Biosci.* 2005; 30(3):303–12.
25. Warren GL, Lowe DA, Hayes DA, Karwoski CJ, Prior BM, Armstrong RB. Excitation failure in eccentric contraction-induced injury of mouse soleus muscle. *J. Physiol.* 1993; 468:487–99.