When Active Muscles Lengthen: Properties and Consequences of Eccentric Contractions

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When the force applied to a muscle exceeds the force produced by the muscle it will lengthen, absorbing mechanical energy. These eccentric contractions, which result in both braking and storing elastic recoil energy in normal locomotion, require very little metabolic energy, yet they are characterized by high force production.

Any time the magnitude of the force applied to a muscle exceeds that produced by the muscle, it will lengthen. Lengthening, or eccentric, muscle contractions have a surprisingly long history in physiological studies. (Note that this word was first introduced as "excentric" by Asmussen in 1953. This original spelling is more enlightening as it combines the prefix ex, "from or away," with centric, "center," hence a muscle contraction that is moving away from the muscle's center.) In 1882, Fick observed that a muscle could exert greater force when stretched while contracting. Fifty years later, Hill reported another feature of eccentric contractions, namely that there is decreased energy liberation in a muscle that is stretched during a contraction. But the first revelation of the functional significance of these properties occurred by way of a clever demonstration devised by Bud Abbott, Brenda Bigland, and Murdoch Ritchie (1). They connected two stationary cycle ergometers back-to-back with a single chain, such that one cyclist pedaled forward and the other resisted this forward motion by braking the backward-moving pedals. Because the internal resistance of the device was low, the same force was being applied by both individuals, yet the task was much easier for the individual braking. This demonstration cleverly revealed that a tiny female resisting the movement of the pedals (in this case, Bigland) could easily exert more force than, and hence control the power output of, a large burly male pedaling forward (Ritchie).

However intriguing, relatively little more was done to probe the properties of lengthening muscle contractions. Conventional wisdom continued to focus on work done by shortening muscles as essential during locomotion. Furthermore, most of the classic studies in muscle physiology, which have formed the foundation of our basic understanding of how muscle works, are founded on two important experimental approaches: isometric (constant length) and isotonic (shortening against a constant load) muscle contractions. As a consequence, much less is known of both the mechanics and the energetics of activated muscle during forced lengthening than during shortening or remaining at a fixed length. In fact, so little is known that the late muscle biomechanist Tom McMahon and his student Jason Harry characterized lengthening contractions as "the dark side of the force-velocity curve"; a reference to the relative lack of knowledge about this region of the classic model of Hill that describes the relationship between a muscle's shortening velocity and its force production. Only recently are both the importance and prevalence of lengthening contractions in normal locomotion receiving increasing attention (see references in Ref. 5).

Muscles as compressible shock absorbers

When the force exerted on the muscle exceeds the force developed by the muscle, work is done on the stretching muscle and in the process the muscle absorbs mechanical energy. This is often referred to as the muscle doing "negative work" (1). What becomes of that absorbed energy depends on how the muscle is being used. The energy can be dissipated as heat, in which case the muscle is functioning as a damper or shock absorber. When hiking downhill, especially a steep hill, this is the primary function of the locomotor muscles (Fig. 1). For example, a 70-kg person descending 500 m absorbs ~350 kJ of energy, enough energy to increase body temperature by 4–5°C. However, the energy absorbed during locomotion can also be stored temporarily as elastic recoil potential energy and subsequently recovered. For example, when running, kinetic energy is absorbed each time the foot hits the ground and continues to be until the center of mass passes over the foot, the point at which both the gravitational potential energy and kinetic energy are at their minima (hence elastic recoil potential energy is at its maximum) during a normal running stride (see, e.g., Refs. 3, 5, and 6). A large portion of this absorbed energy is recoverable, adding to the active force produced on the subsequent stride. For example, during running, trotting, hopping, and jumping the muscle-tendon system functions as a spring when the muscle lengthens while activated, before subsequently shortening (Fig. 1). This stretch-shortening cycle results in improved running economy by a significant enhancement of the power output of the subsequent contraction. In vivo measurements demonstrate that this increase in force production may exceed 50% (8, 13).

In this capacity, the muscles and their tendons are behaving as springs that cyclically absorb and recover elastic recoil energy. Significantly, this function is time dependent; if not recovered, this energy too is lost as heat (3). Hence combining both of these important properties (shock absorber and time-dependent spring) into a single model, the muscle is functioning like a shock absorber in series with a spring. This concep-

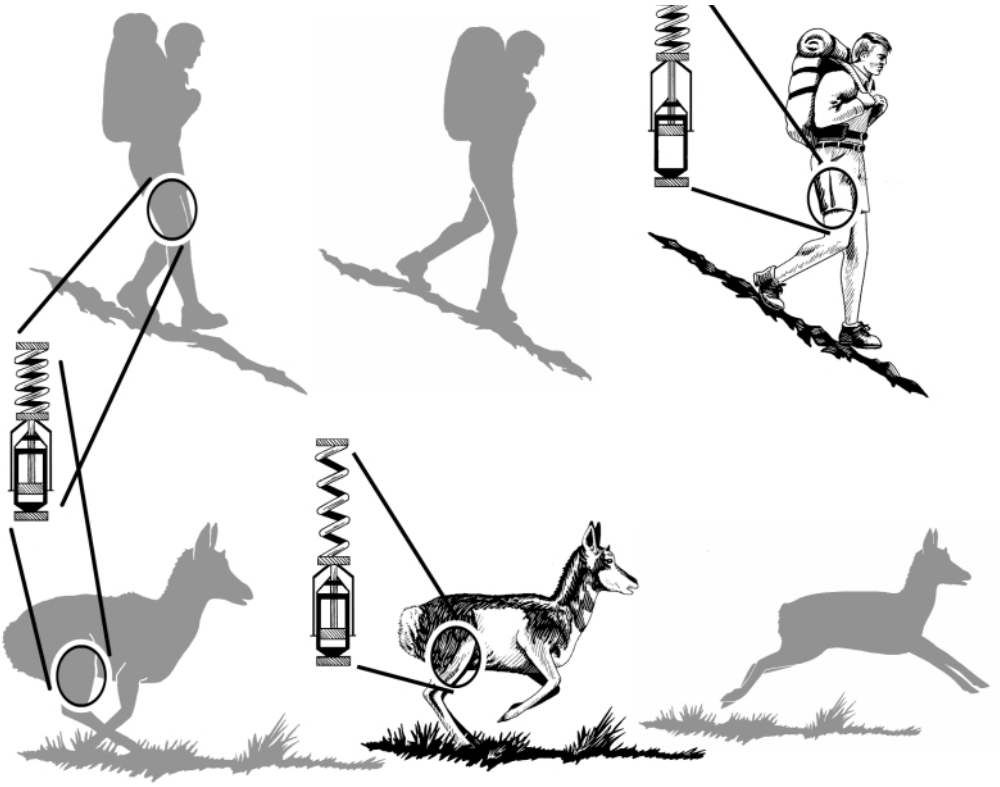

FIGURE 1. A shock absorber functions as a damper when a noncompressible fluid is driven past a piston, converting kinetic energy to heat. If the shock absorber is in series with a spring, then stretching the spring-shock results in tension on the spring or extension of the shock, depending on both the magnitude and time course of the force produced. When an active muscle is lengthened during an eccentric contraction, it behaves like a shock absorber-spring complex. In hiking downhill, nearly all of the energy that stretches the active muscle is lost as heat (extension of the shock). In contrast, running mammals store most of the energy required to stretch the muscle as elastic recoil potential energy (extension of the spring), which can be recovered on the subsequent stride. The time course of stretch and recovery of elastic recoil energy are dependent on both the magnitude of the forces involved as well as the compliance (spring property) of the muscle. As both of these properties are body size dependent, small animals move with predictably higher stride frequencies than do large animals.

tual model captures two features of eccentric contractions: energy absorption and its time-dependent recovery (Fig. 1). Because the spring function is time dependent, any shift in the cycle duration of muscle use, for example stride frequency, will also result in a shift in the fraction of the energy that is recovered vs. that lost as heat, thus impacting the cost of locomotion. While not strictly analogous to a resonating frequency (but see Ref. 6), the time course of muscle stretch and subsequent shortening may be the single most important variable dictating stride frequency among animals; animals should be expected to move by using a stride frequency that will maximize energy recovery. In fact, running mammals spanning three orders of magnitude in body mass have been successfully modeled as a simple mass and spring system. The conclusion of this modeling is that stride frequency among running mammals can be predicted by the relatively simple concept that muscles and their tendons and ligaments function to maximally recover stored elastic energy (6).

A physiology teaching laboratory that we use, inspired by the work of Claire Farley and C. R. Taylor, demonstrates quantitatively both the energy saving and time dependency of muscles functioning as springs that temporarily store and recover elastic recoil energy. When hopping in place, a student volunteer quickly selects a preferred hopping frequency that feels comfortable and is highly reproducible for any given individual. By measuring oxygen uptake, it can be demonstrated that this preferred frequency is the frequency that minimizes the energetic cost per hop. When forced to hop at half this frequency (controlling for hop height, we use a vertical jump height equal to 107% of the subject's eye height for this lab), the cost per hop doubles. This lab demonstrates the striking energy savings that results when elastic recoil supplements the force that the muscles must produce when operating cyclically, in this case against gravity alone; the cost is halved or 50% of the energy is recovered. In addition, this lab demonstrates the body size dependency of this frequency. When the specific preferred frequency is plotted on the graph that describes stride frequency as a function of body mass across a full size range of galloping mammals (15), it falls very close to the value predicted for a mammal of the subject's body size (see Ref. 6). Taylor has made the point that hopping on two feet in a biped is biomechanically similar to galloping in a quadruped. Usually, this lab demonstrates yet another principle of (novel or naïve) eccentric contractions: the subject is almost certain to experience some muscle soreness the next day. If hoping frequency is set solely to maximize elastic recoil in humans, perhaps stride frequency is chosen to maximize energy recovery in all mammals. This may also explain why, within any gait, animals select a relatively constant stride frequency and change speed primarily by changing stride length. It would be energetically

costly to deviate from this preferred, energy-recovering frequency. Muscles can recover 50% (or more) of the energy that would otherwise be lost by simply "tuning" the frequency of their use.

Adaptability of the muscle spring

If the "spring property" of muscle is crucial for energy saving, one might speculate that shifts in demand (i.e., the pattern or nature of muscle use) might result in alterations in the muscle's spring properties. In other words, perhaps this muscle characteristic, like the contractile and metabolic properties of muscle, is also phenotypically plastic. If a muscle is chronically subjected to an eccentric load, does it respond with increased stiffness of the muscle spring? One might expect that a stiffer spring could have two impacts. First, it could act to protect the stretching muscle from stretch overload damage. For example, someone unaccustomed to hiking downhill is likely to experience delayed onset muscle soreness from a single downhill hike (or being a subject in the hopping lab), whereas anyone who hikes downhill regularly has no discomfort whatsoever (in response to a stiffer spring). Second, a stiffer spring could enhance the amount of elastic recoil energy available in the stretch-shortening cycle (8). Seyforth et al. (13) have demonstrated the importance of this enhancement in a task such as a long jump. Could a stiffening of the muscle spring result in greater net force during this kind of activity? We have used two different models to examine these possibilities.

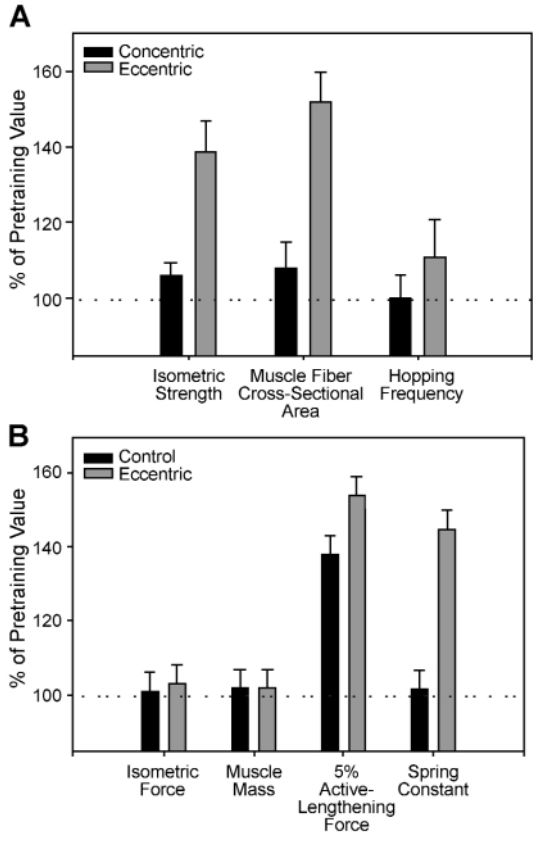

FIGURE 2. Chronic eccentric training results in shifts in muscle properties in both humans and rats. *A*: after 8 wk of chronic eccentric training on an eccentric ergometer, muscle fiber cross-sectional area, isometric strength, and hopping frequency all increased significantly in humans. *B*: when rats are run downhill with supplemental weights equal to 15% of body weight, the result is (despite no significant increase in muscle strength) a significant increase in the spring stiffness (measured as added force resulting from a lengthening contraction). In this preparation, the tendon was bypassed; hence all of the enhanced force production was attributable to muscle only (9, 11).

We have developed an eccentric cycle ergometer that uses a 3-hp motor to drive the pedals in a backward direction. In resisting or slowing this pedal movement, the subject experiences eccentric contractions of the knee (quadriceps) and hip extensors (hamstrings and gluteals). We found that when the workload was increased slowly (in both intensity and duration) over several days, previously sedentary subjects experienced no muscle injury (i.e., no loss of muscle strength), little discomfort (10), and after 8 wk were producing force eccentrically during their 1/2-h daily training periods that exceeded in magnitude the current 1-h cycling record if it had been produced concentrically. Following 8 wk of training, both muscle strength (measured isometrically) and apparent cross-sectional area (of biopsied muscle fibers) increased by ~40% (Fig. 2*A*). In contrast, subjects exercising at the same metabolic exercise intensity (measured as exercise heart rate) on a standard ergometer had no change in either muscle strength or size (9). Furthermore, this activity apparently resulted in a stiffening of the "muscle spring." All of the subjects in this study selected a higher preferred hopping frequency at the conclusion of 8 wk of chronic eccentric training (Fig. 2*A*). In fact, the 11% increase in hopping frequency ($P = 0.001$) is equivalent to the increase in frequency predicted for a 50% reduction in body size among mammals (15). Thus not only did eccentric training result in an apparent protection from muscle damage (which would have been severe in naïve subjects exercising at this high intensity), but, significantly, there was a shift in the muscles' fundamental spring property. This stiffer spring manifests itself with a change in the preferred hopping frequency.

To probe the nature and cause of this shift, we have employed an animal model (11). With only positive reinforcement, rats were easily taught to move downhill on a treadmill while wearing small "backpacks" that held an additional 15% of each animal's body mass. This weighted downhill model represents a significant eccentric load, because the rats must use their locomotor muscles for braking. After 8 wk of downhill locomotion, we measured both the isometric force produced by the triceps muscles as well as the increment increase in that force when the active muscle was stretched (i.e., when the muscle is stretched during an isometric contraction). A lengthening of just <2% of the muscle length (0.58 mm in a muscle 30 mm long) increased the force production by ~38% in a control (nontrained) muscle, whereas the identical stretch in eccentrically trained animals resulted in an enhancement of force equal to 54% over the control isometric (despite no increase in muscle size; Fig. 2*B*; Ref. 11). This large increase in the "dynamic stiffness" of the muscle could be useful not only for increasing the magnitude of elastic recoil energy recovered per stride, it could also be important in protecting the muscle against eccentric injury.

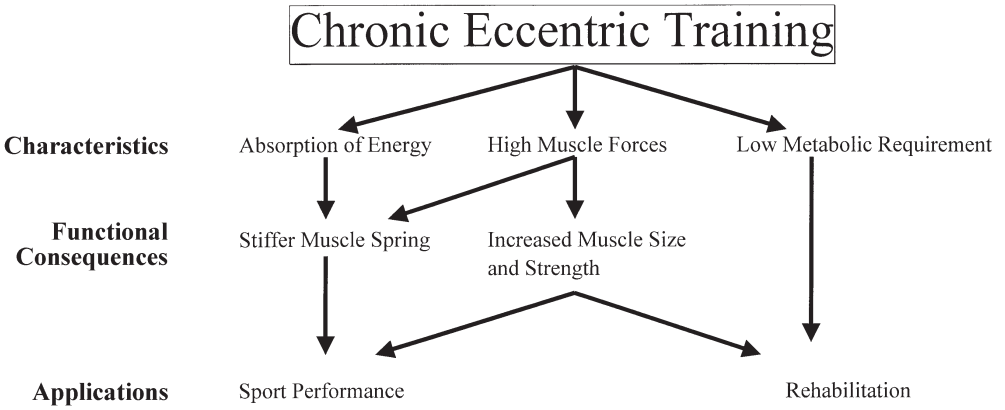

FIGURE 3. Chronic eccentric training triggers a suite of muscle adaptations that have significant and desirable effects in both rehabilitation and sports.

Eccentrics: high force can cause muscle injury

Because muscle produces force, any substantial shift in the normal pattern of muscle use may result in muscle soreness if either the nature or the magnitude of the force production changes significantly (for example, a new exercise or novel repeated task, etc.). Since muscle damage induced by exercise is such a common phenomenon, the mechanisms responsible for damage, recovery, and prevention have received a great deal of attention. In identifying the responses of muscle to damage and injury, these studies have contributed greatly to the process of understanding muscle regeneration and how muscle can be protected from damage. The most notorious symptom after unaccustomed activity is a delayed onset of pain, which is usually accompanied by the presence in the serum of intracellular muscle enzymes or proteins, suggesting damaged fibers (references in Ref. 12). The key functional change, which provides the confirmation of impaired fibers and hence muscle injury, is a decreased ability to produce muscular force.

Perhaps because of the strong association of eccentric contractions and muscle damage/injury, chronic eccentric training has seldom been attempted experimentally. The norm in science seems to be that much less evidence is required to establish an idea as a "fact" than is required to dislodge an idea once established (the "sufficiency of proof" axiom). Once accepted, any observed cause-effect relationship becomes the paradigm within which future experiments are designed and interpreted. In fact, we have learned a great deal about how muscle responds to damage/injury through this valuable model of high-force, acute eccentric contractions. However, it is essential to note that although eccentric contractions can and often do result in muscle damage/injury, eccentric contractions need not cause any muscle damage or injury whatsoever. Unfortunately, the notion linking eccentric contractions and muscle damage/injury persists and likely accounts for the dearth of chronic eccentric training studies.

Just as any novel task can result in muscle soreness, regularly repeating that task usually results in specific muscle accommodations that function to protect against damage or even soreness. It may not be eccentrics per se that are damaging but rather that muscle damage results from exposure to any high-force, novel muscle task. Hence, when eccentric contractions are low force initially and increased in both force and duration slowly over time, no injury occurs. Proposed explanations for the apparent protective effect of repeated eccentrics ("the repeated-bout effect") include elimination of weak areas of certain muscle fibers following an initial exercise bout, changes in the recruitment of motor units with subsequent exposures to eccentric contractions, and the formation of a more resilient muscle structure (12, 14). We speculate that stiffening of the muscle spring, though its exact nature may be poorly described (see below), must also contribute significantly. What is certain, however, is that muscle injury is not a necessary prerequisite for these protective adaptations to occur.

Eccentrics in rehabilitation and sport

Chronic eccentric exercise is characterized by a unique suite of attributes that result in several functional modifications to muscle. Collectively, these changes may have profound applications to patient populations and/or to those interested in enhancing sport performance (Fig. 3).

Because much greater force can be produced eccentrically than concentrically, it has the capability of "overloading" the muscle, the goal of resistance strength training. Force of this magnitude (in excess of the maximum isometric force) is only possible during eccentric (vs. isometric or concentric) contractions. However, not all eccentric contractions result in high loads. If an exercise is designed to simply recover eccentrically the forces generated concentrically, then that exercise does not take advantage of this unique property. It is the electric motor of the eccentric cycle ergometry that is generating the high forces that the muscles oppose; these greatly exceed in magnitude the muscular force that could be generated concentrically.

Furthermore, because eccentric muscle contractions occur with very little metabolic cost, muscles contracting eccentrically produce "more for less"; they generate high mechanical muscle tensions at low metabolic costs. Eccentric contractions not only produce the highest forces but do so at a greatly

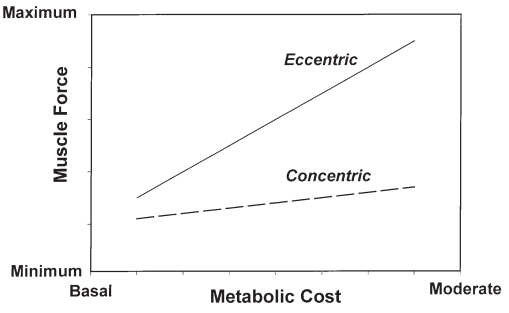

FIGURE 4. Bigland-Ritchie (2) first plotted oxygen uptake against work showing a 6- to 7-times reduction in metabolic cost to achieve the same magnitude of work (negative vs. positive). If we reverse the axes, it is apparent that at identical exercise intensities (measured as oxygen uptake) much more force can be generated eccentrically than concentrically at a moderate metabolic cost.

reduced oxygen requirement; an observation first documented by the pioneering work of Bigland-Ritchie et al. (2), who reported that the oxygen requirement of submaximal eccentric cycling is only 1/6–1/7 of that for concentric cycling at the same workload.

Thus eccentric training can increase the size and strength of locomotor muscle (Fig. 2*A*) with very little demand on the cardiovascular system (9). The magnitude of observed increases in both strength and fiber area with eccentric training often exceeds that seen following a similar duration of traditional resistance strength training. With chronic eccentric training there is also a resultant stiffening of the muscle spring that can occur independent of, or in addition to, increases in size and isometric strength of the muscle (11). In all probability, the force enhancement and spring changes following chronic eccentric training are likely due to both structural and neural influences.

Application to rehabilitation and sport

The potential application of chronic eccentric exercise to the elderly and patients suffering from diseases that limit either the uptake or delivery of oxygen, e.g., chronic obstructive pulmonary disease or chronic heart failure, is alluring. These individuals may be so severely exercise limited that walking may be at or beyond their aerobic capacities, eliminating exercise at intensities sufficient to prevent muscle wasting (sarcopenia). Any exercise that requires a significant increase in ventilation and cardiac output may be not only uncomfortable but for many elderly impossible. Therefore, chronic eccentric training may be a high-force yet nonstrenuous (low metabolic cost) rehabilitation countermeasure with the potential to overcome these skeletal muscle deficits and diminished ability to function independently (Fig. 4). In addition, as a result of the increased stiffness (tighter muscular spring) in muscle following eccentric exercise, there may be improvements in sport performance activities, such as jumping (13). In a preliminary study comparing basketball players trained for 6 wk with either high-force eccentric cycle ergometry or with a traditional strength/power resistance program, we noted increases in vertical jumping height in excess of 8% in the eccentrically trained group, whereas those in a traditional resistance training group showed no change in jump height.

Where is the muscle spring?

Because tendon was historically thought to be the source of passive tension, the production of passive tension and storage and release of elastic recoil energy in skeletal muscle had been considered negligible (16). However, coupling the physiology of lengthening muscles with the emergent knowledge of the cytoskeletal proteins within the muscle cell, it is becoming clear that these proteins contribute greatly to the storage of elastic energy.

Likewise, intact fiber elasticity and passive tension production were thought to reside predominantly in the extracellular collagen matrix. However, within the physiological limits of stretch most of the passive tension produced by a myofibril is due to the elastic titin filament, whereas collagens and intermediate filaments become important only with further stretch (see references in Ref. 4).

Although normally studied under passive stretch, perhaps titin contributes significantly to the production of active tension when the muscle actively lengthens in resisting an external load. Titin may contribute by enhancing elastic energy storage and release and by maintaining sarcomere alignment, ensuring efficient muscle contraction (16). Quantitative gel electrophoretic mobility studies on SDS gels as well as recent molecular studies have identified the existence of several isoforms within the I band region of titin. Because there is only one titin gene, titin isoforms are generated by the differential splicing of the elastic region of the molecule. It is thought that the expression of different titin isoforms could adjust the spring properties of the fiber to the physiological demands placed on the muscle to best maintain sarcomere structure (see references in Ref. 7). A stiffer, shorter titin, therefore, may explain the greater passive (spring constant) as well as active lengthening force-producing capabilities following eccentric training.

Is there evidence supporting titin as a major contributor to the muscle spring? We can compare quantitatively titin's potential contribution with the measured stretch potentiation of the triceps muscle. By use of laser tweezers, the maximum force has been estimated for individual titin molecules; published values range from 5 to 70 pN. In estimating the possible force that could be attributable to titin, we make the following assumptions: *1*) there are thought to be six titin molecules per half myosin filament; *2*) there are 7×10^8 thick filaments per square millimeter of myofibril; and *3*) we calculated the cross-sectional area of the triceps muscle of these rats to be 35 mm^2. Together, we estimate the total force attributable to the stretch of titin to be

$$(5\text{–}70\ pN/titin^{-1}) \times (6\ titins/thick\ filament) \times (7 \times 10^8\ thick/mm^2) \times (35 mm^2/cross\ section)$$

or a range of 0.74–10.3 N, which includes the range of increased tension that we measured in the rat triceps muscle of 5–7 N, resulting from a stretch of a muscle under tetanic contraction. Because this is a pinnate muscle, the functional cross-sectional area may be considerably higher, hence the maxi-

mum force attributable to titin may even exceed our estimated values.

Titin's role in lengthening contractions may also include the initiation of cellular signaling to enhance cross-bridge recruitment while decreasing the cost. Hence, a differential expression of titin isoforms could alter the magnitude of elastic recoil storage and subsequent utilization as well as effect cross-bridge cycling and efficiency.

Summary and conclusions

Our view of muscle is usually that of a tension-producing machine that, when shortening, provides the work necessary for organisms to move about. However, movement also requires that muscles function to absorb kinetic energy, fluctuations that are inevitable during locomotion. Any time the force acting on the muscle exceeds the force produced by the muscle, the muscle will lengthen while producing force. During normal locomotion, these eccentric contractions function in two capacities: *1*) they dissipate absorbed energy as heat, to function as a damper or shock absorber, reducing the kinetic energy via braking, and *2*) conversely, the energy absorbed in stretched muscle (and tendon) may be stored as elastic recoil potential energy and subsequently recovered, allowing the muscle to effectively function as a spring. This "spring property" of muscle is both time dependent, which may be the rule that sets stride frequency among mammals, and adaptable, the muscle spring becoming stiffer in response to chronic eccentric loading. Two unique properties of eccentric contractions are physiologically fundamental. The energy cost for eccentric contractions is unusually low and the magnitude of the force produced is unusually high. As a consequence, muscles exposed to chronic eccentric training respond with significant increases in strength and size as well as alterations in the spring properties of the muscle. These predictable responses have both clinical and physical performance consequences. Loss of muscle mass and strength are thought to be nearly inevitable consequences of aging, accelerated by both heart and respiratory disease. Chronic eccentric exercise, which requires minimal energy and thus oxygen support, may be ideally suited for both rehabilitation for this population as well as increasing both strength and power in all individuals. Finally, while the muscle spring properties are often attributed to collagen and tendons (structures outside the muscle fiber), evidence suggests that the gigantic protein titin may contribute significantly to these important and adaptable functions of skeletal muscle inside the fiber.

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