

# Elastic Properties of Active Muscle — On the Rebound?

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MONROY, J.A., A.K. LAPPIN, and K.C. NISHIKAWA. Elastic properties of active muscle — on the rebound? *Exerc. Sport Sci. Rev.*, Vol. 35, No. 4, pp. 174–179, 2007. *During active lengthening and shortening, muscles exhibit a variety of time-dependent spring properties, including load-dependent and nonlinear stiffness. These properties can be explained as interactions between a spring element and cycling cross bridges within muscle sarcomeres. Several lines of evidence suggest a role for the giant protein titin in active muscle, but specific mechanisms remain to be elucidated.* **Key Words:** elastic recoil, force-velocity curve, series elastic elements, stiffness, striated muscle, titin, viscoelastic properties

## INTRODUCTION

Animals are remarkable in the extremes of speed and dynamic stability that they achieve during movement (23). During natural movements, muscles exhibit a variety of functions, serving not only as motors but also as springs, brakes, and struts (9). Our understanding of the mechanisms of muscle contraction are based on a remarkably limited set of laboratory conditions, such as isometric tetanus, isotonic shortening, and rigor. It is surprising how little is known about the mechanical functions of muscle under natural conditions of activation and external loading (20,23). The purpose of this review is to examine muscle contraction during active lengthening and shortening from the theoretical perspective of viscoelasticity or the behavior of damped springs.

We first provide a brief history of the spring properties of active muscle. We next review Hill's force-velocity relationship (14) and examine a common misconception that has resulted from applying this paradigm to animal movements. We next review studies of elastic recoil in actively shortening muscle. We show how the nonlinear load-dependent stiffness of muscles during elastic recoil confers self-stabilization to perturbations in load, and we develop a general model that describes the stiffness of muscle during

isometric force development, isotonic shortening, and rapid unloading. We next propose that history-dependent properties, including depression of force with shortening and enhancement of force with stretch, match predictions for the behavior of a damped and time-dependent spring. These considerations lead us to postulate the existence of a spring within active muscle sarcomeres that does not interact cyclically with the thin filament. The giant elastic protein titin is ideally suited to serve such a role. Although a role for titin in active muscle sarcomeres is often postulated, it remains to be demonstrated definitively.

## SPRING PROPERTIES OF ACTIVE MUSCLE: HISTORY OF THE CONCEPTS

In his 1953 article on the “instantaneous elasticity” of active muscle, A.V. Hill (15) wrote “The central thesis of this paper is that the process of muscular contraction, as such, has nothing to do with elasticity. It is true that muscles have elastic properties, though the most obvious of these clearly reside in the inert elastic material, which lies in series, or in parallel, with the contractile substance, while the elastic qualities of resting muscle are well known. During activity, however, it is hard to specify any mechanical property of contractile substance which can usefully be attributed to elasticity.” Thus, Hill believed that the spring properties of active muscle arise from an interaction between active nonspringy motors (his “contractile substance”) and inert springs arranged in series or parallel with the contractile substance. Springs in series with a muscle are stretched when the muscle contracts (*e.g.*, tendons), whereas springs in parallel with a muscle are stretched when the muscle is stretched (*e.g.*, sarcolemma or epimysium).

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With the advent of the sliding filament theory (16,18), Hill's dichotomy between active contractile elements versus inert elastic elements remained relatively unchanged. Although active muscle was shown to exhibit viscoelastic properties, these were attributed solely to the cross bridges because the viscoelastic behavior of stretched muscle fibers varied in direct proportion to the amount of overlap between the thick and thin filaments (17). The viscoelastic behavior of the cross bridges continues to be an area of active research (24).

Despite the huge success of the sliding filament theory, it has proved insufficient to explain several springlike features of muscle function, including enhancement of force with stretch, depression of force with shortening, and the low cost of force production during active stretch. At the level of the whole organism, it is clear that these springlike properties of active muscle play an important role in most activities (9,21).

## THE FORCE-VELOCITY RELATIONSHIP

Muscles themselves have been thought to contribute little to the power of fast movements because they shorten rapidly only under very low loads (2). This idea follows directly from Hill's force-velocity curve (14). Because power is the product of force and velocity, the force-velocity property of muscle necessarily limits power output. When the force is large, the velocity is small, and when the force is small, the velocity is large (Fig. 1C).

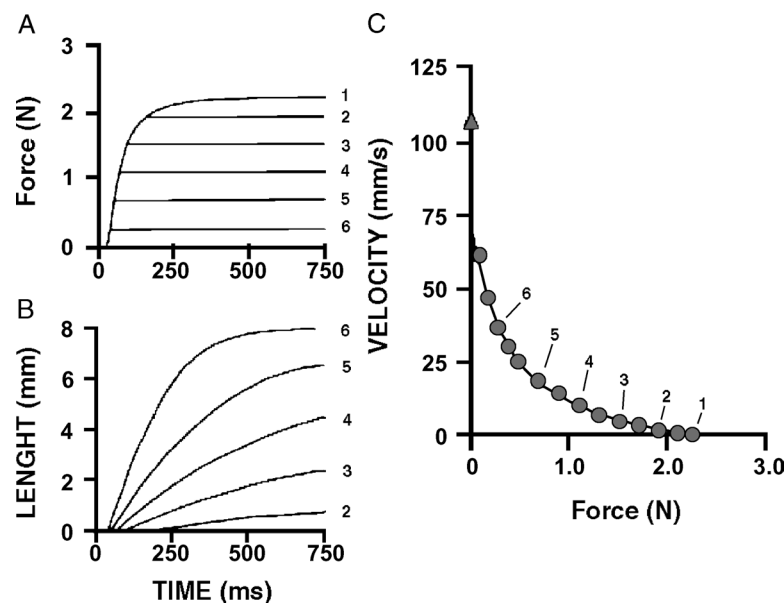
The apparent trade-off between force and velocity is, at least in part, an artifact of Hill's after-loaded isotonic paradigm for generating the force-velocity curve (Fig. 1). In Hill's after-loaded paradigm (14), as the external load

increases, the duration of muscle stimulation before the onset of shortening also increases (Fig. 1A). At the smallest loads, a muscle is stimulated for a very short duration (as little as 10–15 milliseconds), whereas at the largest loads, a muscle is stimulated for much longer durations (>250 milliseconds). Hill matched the duration of stimulation to the afterload specifically to remove any contribution of muscle series elasticity to the observed shortening velocity, to isolate the properties of the contractile apparatus (14).

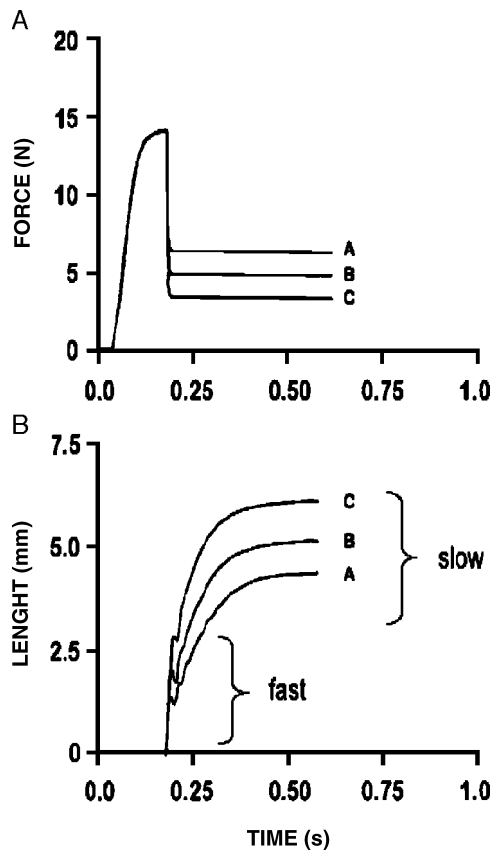
Natural movements rarely, if ever, fall within the constraints on stimulation and load that obtain for the after-loaded isotonic paradigm. In fact, we expect that animals should avoid activation patterns that produce isotonic shortening because they minimize power output. When muscles contract against a resisting force, they typically are activated for a relatively long duration, after which, they shorten upon unloading. This occurs, for example, when we slide our thumb across our index finger to produce a snapping sound or when our ventricles contract against the pressure of venous blood entering from the atria. During shortening, elastic elements both inside and outside the muscle recoil elastically, enhancing the velocity and power of the resulting movement (20).

## ELASTIC RECOIL OF ACTIVELY SHORTENING MUSCLES

When a muscle contracts against a resisting force, it will recoil elastically when the force-resisting movement is reduced. Even a skinned single muscle fiber exhibits this behavior (25). The load-clamp test (Fig. 2) can be used to investigate elastic recoil of muscle during active shortening. During a load-clamp test, the load is reduced rapidly after a



**Figure 1.** Hill's after-loaded isotonic paradigm for generating the force-velocity curve. A muscle is stimulated and contracts isometrically until it reaches a force at which it can move the afterload. Note that the duration of stimulation before the onset of shortening increases with the afterload. Force (A) and change in length (B) records for six afterloads are shown. The corresponding points on the force-velocity curve are shown in (C). [Adapted from Caiozzo, V.J. Plasticity of skeletal muscle phenotype: mechanical consequences. *Muscle Nerve* 26:740–768, 2002. Copyright © 2002 John Wiley and Sons. Used with permission.]



**Figure 2.** Load-clamp technique for investigating elastic recoil of active muscle. A muscle is fully activated (A). After 200 milliseconds, the load is rapidly reduced. The resulting change in length is biphasic. In the initial fast phase, the muscle shortens rapidly because of unloading of elastic elements (B). This is followed by a slow phase of shortening. Three different loads are shown. [Adapted from Caiozzo, V.J. Plasticity of skeletal muscle phenotype: mechanical consequences. *Muscle Nerve* 26:740–768, 2002. Copyright © 2002 John Wiley and Sons. Used with permission.]

period of isometric stimulation (Fig. 2A). When the load is reduced, an initial rapid change in length due to recoil of elastic elements is followed by a slower change in length due to cyclic interactions of contractile proteins within the muscle (Fig. 2B). The shortening velocity during the initial rapid phase may greatly exceed  $V_{max}$ .

Recent studies demonstrate that when muscles recoil elastically, they behave as nonlinear load-dependent springs (20). As a muscle develops force isometrically, elastic elements within the muscle are strained from their equilibrium length. When the resisting force is reduced, the elastic elements recoil to their resting length. The displacement of elastic elements during recoil increases exponentially with the change in load (Fig. 3A). Based on this observation, a simple model of elastic recoil in active muscle was developed (20). The model predicts the displacement of the muscle spring ( $x_m$ ) as a function of the change in force during unloading ( $\Delta F$ ):

$$x_m = 10^{(\Delta F - c_1)/c_2} \quad (1)$$

The shape of the exponential functions is described by two arbitrary constants,  $c_1$  and  $c_2$ , which increase linearly with the force that develops before unloading (Fig. 3B).

These constants seem to be invariant over a wide range of muscles and species (22).

The spring constant ( $k_m$ )  $\text{Nm}^{-1}$  is given by the first derivative of the inverse of equation 1 [ $\Delta F = c_1 + c_2 \log(x_m)$ ]:

$$k_m = c_2 / (2.303x_m) \quad (2)$$

During elastic recoil, muscle stiffness decreases rapidly and nonlinearly (Fig. 3A) as the change in force during unloading ( $\Delta F$ ) increases (20). For a given external load, stiffness decreases with increasing force before unloading as the change in force during unloading increases (Fig. 4). Therefore, because mass increases with the cube of length but force increases with the square of length, the stiffness of actively shortening muscles during elastic recoil will be higher in smaller animals than in larger animals. The stiffness of actively shortening muscles will also be higher in older sarcopenic animals than in younger animals with a larger cross-sectional area of muscle.

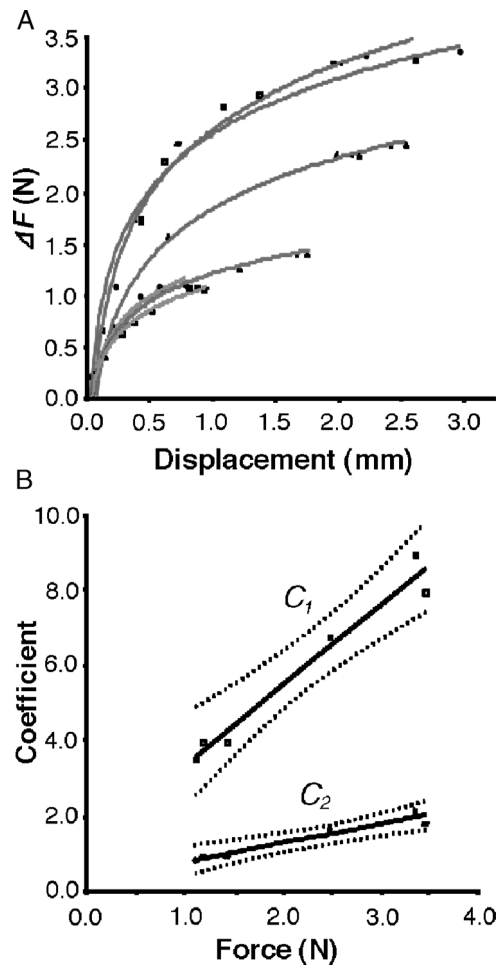
The nonlinear load-dependent stiffness of muscle during active shortening gives rise to intrinsic self-stabilization to perturbations in load (Fig. 4). For a given force before unloading, a muscle will become stiffer when the external load increases (upward arrow) and will become more compliant when the load decreases (downward arrow). When the load changes unexpectedly, the muscle itself has the capacity to adjust its stiffness rapidly without requiring neural input (20).

It has been suggested (20) that this nonlinear load-dependent stiffness of muscle during elastic recoil could be explained if the cycling cross bridges impose a strain on elastic elements within a sarcomere during isometric force development. Load-dependent stiffness during active shortening would result from the interaction between strained elastic elements and the load-dependent cycling of the cross bridges within a sarcomere (14). In contrast, because elastic elements outside the sarcomere (*i.e.*, epimysial, myofascial, and myotendoninous connective tissues) cannot interact directly with the cycling cross-bridges, they do not exhibit nonlinear load-dependent elastic properties (20,30). In contrast, their stiffness is relatively constant over the physiological range of force (2,20).

## HELPING THE TWAIN TO MEET

In contrast to the load-clamp studies described above, numerous studies have established that muscle stiffness changes linearly with force during both isometric tetanus (3) and isotonic shortening (29). How can we reconcile these observations with the nonlinear load-dependent stiffness of muscle during elastic recoil?

We used the elastic recoil model (20) to estimate the elastic modulus of frog skeletal muscle under a variety of conditions, including isometric tetanus and isotonic shortening, in addition to elastic recoil (Fig. 5). The elastic recoil model predicts a linear increase in elastic modulus with force during both isometric tetanus (solid black line) and isotonic shortening (dashed black line). The model predicts an elastic modulus of  $9.5 \times 10^7 \text{ Nm}^{-2}$  for frog depressor mandibulae



**Figure 3.** Elastic properties of skeletal muscle during elastic recoil. Change in force ( $\Delta F$ ) versus muscle displacement ( $x_m$ ) during load-clamp experiments (A): 200-millisecond muscle stimulation before unloading; 50-millisecond muscle stimulation prior unloading; and 100-millisecond muscle stimulation before unloading. Linear regression of coefficients  $c_1$  (N) and  $c_2$  ( $\text{kgs}^{-2}$ ) with muscle force (N) before unloading (B). The coefficients describe the shape of the exponential function relating  $x_m$  to  $\Delta F$  (equation 1). Dotted lines show 95% confidence intervals for the regression slopes. [Adapted from Lappin, A.K., J.A. Monroy, J.Q. Pilarski, E.D. Zepnewski, D.J. Pierotti, and K.C. Nishikawa. Storage and recovery of elastic potential energy powers ballistic prey capture in toads. *J. Exp. Biol.* 209:2535–2553, 2006. Copyright © 2006 The Company of Biologists. Used with permission.]

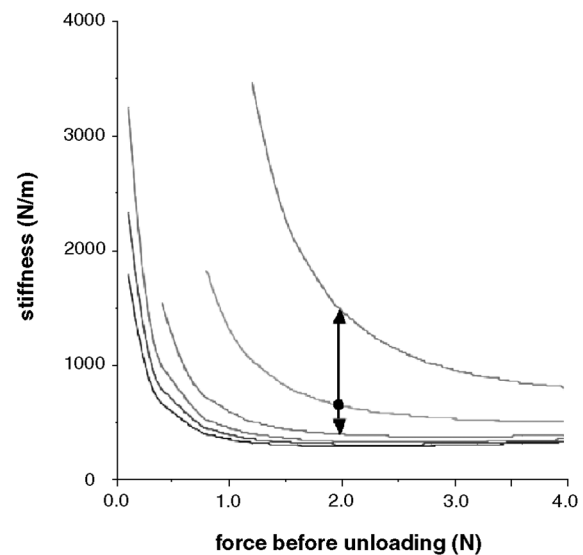
during isometric tetanus at 24–25°C. The stiffness of bullfrog semitendinosus muscle during isometric tetanus is  $6.5 \pm 1.3 \times 10^7 \text{ Nm}^{-2}$  at 19–20°C (12). Given the temperature difference (12), the correspondence between measured and modeled stiffness is relatively close. These results demonstrate the generality of the elastic recoil model, which predicts muscle stiffness not only during elastic recoil but also during isometric tetanus and isotonic shortening.

### HISTORY-DEPENDENT PROPERTIES OF ACTIVE MUSCLE

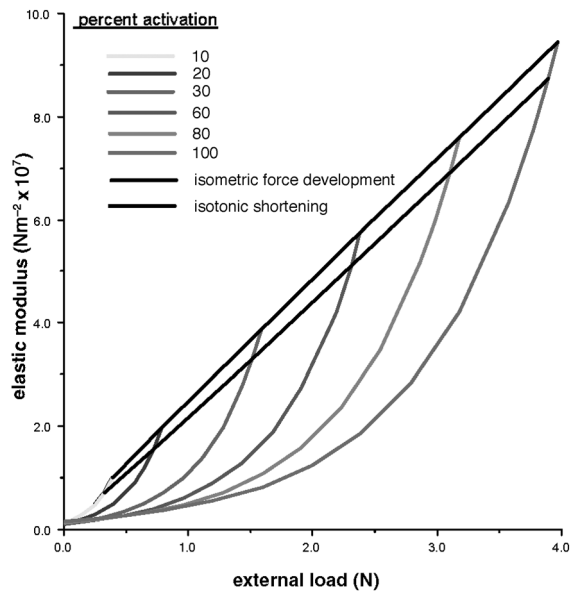
History-dependent changes in active force production include depression of force with shortening and enhance-

ment of force with stretch (1,10,11). In response to a decrease in length, muscle force decreases to a steady-state level that depends upon both the amplitude and velocity of shortening (26). The steady-state force produced by muscles after active shortening is less than the isometric force at corresponding lengths (Fig. 6A). Likewise, the steady state force after active lengthening is higher than the isometric force at corresponding lengths (Fig. 6B). These history-dependent properties of active muscle are exactly those expected of springs, which produce greater tensile force when stretched and less tensile force when shortened, in proportion to their change in length. However, few of the theories that have been proposed to explain history-dependent effects in active muscle deal explicitly with spring properties (26).

These history-dependent properties are not easily accommodated within the sliding filament theory (13,26), which predicts that the steady-state force produced by the cycling cross bridges is independent of the preceding conditions (17). Within the framework of the sliding filament theory, mechanisms of force enhancement during active stretch, and mechanisms of force depression during shortening, historically have invoked processes that affect the internal work done by the myosin heads during cross bridge cycling (26; for exceptions, see 13).



**Figure 4.** Nonlinear load-dependent stiffness ( $\text{Nm}^{-1}$ ) of muscle during elastic recoil as a function of the force before unloading (N). Lines illustrate different loads:  $0.0022 P_0$ ,  $0.0011 P_0$ ,  $0.022 P_0$ ,  $0.045 P_0$ ,  $0.11 P_0$ , and  $0.22 P_0$ . When the load increases, the muscle becomes stiffer. When the load decreases, the muscle becomes more compliant. For example, if a muscle develops a force of 2 N before shortening, its spring constant will be  $648 \text{ Nm}^{-1}$  when shortening against an external load of 0.45 N (dot). If the load increases unexpectedly to 0.9 N (upward arrow), the spring constant will increase rapidly to  $1465 \text{ Nm}^{-1}$ . If the load decreases unexpectedly to 0.18 N (downward arrow), the spring constant will decrease rapidly to  $398 \text{ Nm}^{-1}$ . Change in stiffness with load is an intrinsic property of muscle and requires no input from the central nervous system. [Adapted from Lappin, A.K., J.A. Monroy, J.Q. Pilarski, E.D. Zepnewski, D.J. Pierotti, and K.C. Nishikawa. Storage and recovery of elastic potential energy powers ballistic prey capture in toads. *J. Exp. Biol.* 209:2535–2553, 2006. Copyright © 2006 The Company of Biologists. Used with permission.]



**Figure 5.** Elastic modulus ( $\text{Nm}^{-2}$ ) of frog skeletal muscle under a variety of conditions (see equations 1 and 2) predicted using the elastic recoil model of Lappin *et al.* (20). During isometric force development, the force before unloading ranges from 0.1 to 1.0  $P_0$  (0.4–4 N), and the change in force ( $\Delta F$ ) is zero (solid black line). During isotonic shortening, the force before unloading ranges from 0.1 to 1.0  $P_0$  (0.4–4 N), and the change in force ( $\Delta F$ ) is small ( $<0.1$  N, dashed black line). During rapid unloading, both the force before unloading and the change in force ( $\Delta F$ ) range from 0.1 to 1.0  $P_0$  (0.4–4 N). The model predicts an elastic modulus of  $9.5 \times 10^7 \text{ Nm}^{-2}$  at maximum isometric force and 24–25°C. During isometric force development, stiffness increases linearly with activation (solid line), whereas during isotonic shortening, the stiffness increases linearly with load (dashed line). For a given level of activation ( $P/P_0$ ), the elastic modulus decreases exponentially with the external load during rapid unloading. The elastic modulus converges to a value of approximately  $1.0 \times 10^6 \text{ Nm}^{-2}$  during unloaded shortening, regardless of the level of activation.

Hookean springs change length nearly instantaneously with applied force. Muscles, composed of polymeric proteins in solution, are not expected to respond as quickly. It is well known that muscles exhibit time-dependent changes in force in response to applied changes in length (26). In response to an increase in length, active muscle force increases rapidly to values up to nearly twice the maximum isometric force. The force then decays rapidly to a steady-state value that increases with the amplitude of the stretch and with sarcomere length. In response to a decrease in length, muscle force decreases rapidly and then returns more slowly to a steady-state level that depends upon the amplitude of shortening and the speed of shortening.

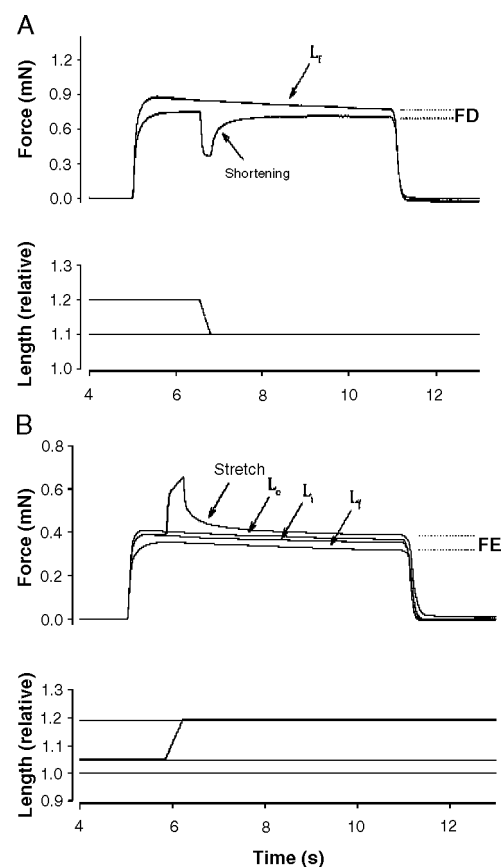
Differences between force enhancement with stretch and force depression with shortening (Figs. 6A, B) are expected for time-dependent springs. During lengthening, the force increases rapidly because the delay in the length response increases the “stiffness” of the spring. The force decays as the change in length of the spring catches up with the imposed length change, and the increase in steady-state force is due to the increased displacement of the spring from its equilibrium position at the new length. During shortening, the delay in the length response of the spring has the opposite effect. The velocity, and hence the kinetic energy,

of a damped spring is greatest at its natural frequency. As the velocity of shortening increases, the degree to which the spring can keep up falls farther and farther behind, so that the amount of force depression actually decreases with shortening speed.

### ADDITIONAL EVIDENCE FOR A NONCROSS BRIDGE SPRING IN ACTIVE MUSCLE

The sliding filament theory predicts that the total active force on the descending limb of the force length curve should never exceed the maximum isometric force at optimum overlap. However, the active steady-state force after muscle stretch on the descending limb of the force-length curve can in fact exceed the maximum isometric force (13,26).

During active stretch, muscles require much less energy to produce a given force than when they contract concentrically (5,21). It seems as though that some of the external work, done in stretching the muscle, can be absorbed to



**Figure 6.** Depression of force with shortening (FD) in a single muscle fiber (A). The fiber was shortened by 10% on the descending limb of the force-length curve at a speed of  $40 \text{ Ls}^{-1}$ . An isometric reference contraction performed at the corresponding final length ( $L_f$ ) is also shown. Enhancement of force with stretch in a single muscle fiber (B). The fiber was stretched by 15% on the descending limb of the force-length curve at a speed of  $40 \text{ Ls}^{-1}$ . In the length traces, 1.0 corresponds to  $L_0$ . Isometric reference contractions performed at the corresponding optimal ( $L_0$ ), initial ( $L_i$ ), and final lengths ( $L_f$ ) are also shown. (Reprinted from Rassier, D.E., and W. Herzog. Considerations on the history dependence of muscle contraction. *J. Appl. Physiol.* 96:419–427, 2004. Copyright © 2004 The American Physiological Society. Used with permission.)

enhance force. It is difficult to explain this observation within the sliding filament theory because elastic energy is stored and recovered within each cross bridge cycle (8), but there is no mechanism for storing energy for more than one cycle. This behavior is exactly as expected for a spring, which stores the work done in stretching it as elastic potential energy.

## CONCLUSIONS

A variety of long-known and well-studied aspects of muscle function are not well accounted for by the sliding filament theory, including enhancement of force with stretch, depression of force with shortening, and the low cost of force production during active lengthening. To this list, we add the nonlinear load-dependent stiffness of muscle during elastic recoil, which confers the significant property of self-stabilization to perturbations in load. All of these properties suggest the existence of a spring within active muscle sarcomeres that interacts with the thin filaments noncyclically and is displaced from equilibrium by the cycling cross bridges during force development.

We hypothesize that elastic elements within muscle sarcomeres play an important role in muscle contraction, not only during active lengthening but also during active shortening (except during isotonic shortening, when the stimulation parameters have been chosen specifically to eliminate their role). Several recent studies have suggested that titin may function as a spring in active muscle (4,7,13,19,21,27,28). Calcium has been shown to increase the tension and stiffness of a noncross bridge structure in active muscle fibers (4,7). Calcium increases titin stiffness, and the force-length relationship of mouse soleus fibers shifts to the left as intracellular calcium concentration increases (19).

Despite these enticing hints, a detailed hypothesis for the role of titin in active muscle contraction awaits development and testing.

## Acknowledgments

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