A Continuous and Differentiable Mechanical Model of Muscle Force and Impedance

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Abstract. No single muscle model exists that has the same mechanical impedance and force development properties as biological muscle. It is essential to develop a muscle model with the same force limitations and impedance as biological muscle, especially for predictive simulations, as these properties are taken into account when choosing a posture for a specific task. We propose a mechanics-based muscle model that has the same impedance and force development properties as biological muscle by making a small topology change that turns titin, an enormous viscoelastic protein, from acting in parallel to the cross-bridges to acting in series with the cross-bridges.

1 Introduction

The stiffness and damping properties of muscle affect not only how the body responds to perturbations, but also the postures that people choose to adopt for a particular task [1]. Unfortunately, no single muscle model captures all of the mechanical properties of muscle: Hill-type muscle models can have a region of negative stiffness on the descending limb of the active-force-length curve [2], Huxley-type models over-estimate the forces developed during rapid eccentric contractions [3], and the active spring-damper muscle models used in motor control simulations [4] have not been extended to include the well-known variation of muscle force with length and velocity. While curve fitting has been used to artificially add the missing stiffness and damping forces to Hill models [5], it is unlikely that this modification will generalize outside of the data used for the curve fit.

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The forces developed by muscle during ramp-stretch experiments provide insight into the mechanisms that underlie stiffness and damping in muscle. When active muscle is stretched, the force increase follows a stereotypical pattern (Fig. 1): a rapid increase in force over a short-range, followed by a more gradual increase over a longer range. As the rate of lengthening is increased, the peak force developed in the short-range increases [6] consistent with a damping element. In contrast, the long-range tension profiles appear to be strictly a function of length: the long-range profiles of Figs. 7A–C of [6] are nearly identical when plotted against muscle length, consistent with an elastic element. Furthermore, the stiffness of the long-range force profile does not appear to change with muscle length nor active force, and therefore is not dependent on the number of attached cross-bridges: Fig. 5A of [7] shows the active force profiles of 8 ramp-stretches of a rat soleus done at lengths ranging from $\tilde{\ell}^{\mathrm{M}} = 0.51 - 0.88$, each with nearly identical long-range force profile slopes. Taken together, the ramp-stretch experiments of [6,7] suggest that the mechanism of force development in lengthening muscle is not due to cross-bridge cycling, but instead due to the lengthening of a viscoelastic element capable of sustaining large strains.

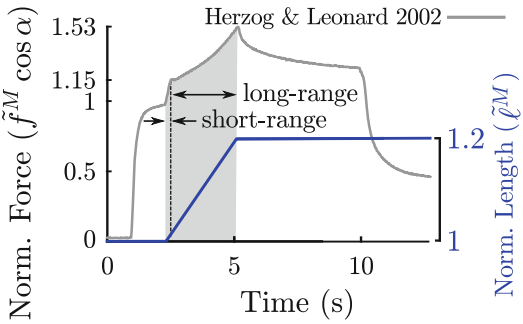

Fig. 1. The stereotypical tension profile of an activated muscle being stretched at a constant velocity. Note that $\tilde{\ell}^{\mathrm{M}}$ is the length of the muscle fiber divided by ℓ_o^{M} the length at which the fiber develops its maximum active isometric force, and $\tilde{f}^{\mathrm{M}}$ is the force of the muscle divided by its maximum active isometric force $f_{\mathrm{o}}^{\mathrm{M}}$.

There is only one known viscoelastic element in a sarcomere capable of sustaining large strains: titin. Titin is enormous, spanning half the length of a sarcomere, is 100× more compliant than either myosin or actin, and develops significant damping forces [8]. The fantastic properties of titin have been proposed to affect active muscle force by attaching to actin, as detailed in the winding-filament theory and realized in a model [9]. Here we present an alternative mechanism which can explain the short and long-range force profiles observed in eccentrically contracting muscle. While it is clear that titin plays a central role in the development of active and passive muscle force, the precise mechanism by which this occurs is not clear.

2 Model

A slight modification to the topology of the sarcomere can change titin from acting in parallel to the cross-bridges (Fig. 2A) to acting in series (Fig. 2B), which provides the active muscle model with titin's stiffness and damping properties. It is known that titin connects to the actin filament of the adjacent sarcomere [10]. Thus if the actin filament can transfer a large fraction of its tension to the titin molecule in the neighboring sarcomere, and is free to move with respect to the Z-line, then it is possible that titin could function in series with myosin (Fig. 2B). We develop a muscle model under the assumption that titin acts in series with myosin (Fig. 2C) as the force response of lengthening muscle bears a striking similarity to that of a spring-damper.

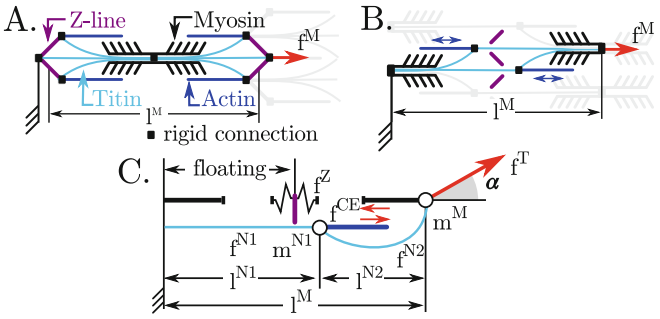

Fig. 2. Although titin is normally treated as a parallel element (A), it may function as a series element (B) since it is connected to the actin filament of the neighboring sarcomere. Assuming symmetry, we model a muscle as a scaled sarcomere that has a series element for titin (C).

To model this new topology we introduce two small masses: one between the titin element and its neighboring actin filament, m^{N1}, and another, m^{M}, between the myosin filament and the applied external force. Since the damping forces of titin scale with its strain [8], we model titin as

$$f^{\mathrm{N}} = f_{\mathrm{o}}^{\mathrm{M}}(\mathbf{f}^{\mathrm{K}}(\ell^{\mathrm{N}}/\ell_{\mathrm{s}}^{\mathrm{N}}) + \mathbf{f}^{\mathrm{D}}(\ell^{\mathrm{N}}/\ell_{\mathrm{s}}^{\mathrm{N}})(\beta v^{\mathrm{N}})) \tag{1}$$

a nonlinear spring-damper where f^{N} is the tension developed by titin, $f_{\mathrm{o}}^{\mathrm{M}}$ is the maximum active isometric force of the muscle, ℓ^{N} is the length of titin, $\ell_{\mathrm{s}}^{\mathrm{N}}$ is the slack-length of titin, v^{N} is the lengthening rate of titin, β is a damping coefficient, $\mathbf{f}^{\mathrm{K}}$ is the normalized passive-force-length curve, and $\mathbf{f}^{\mathrm{D}}$ is a smooth step function that increases from 0 to 1 as a function of ℓ^{N}. The force developed by the cross-bridge cycling between the myosin and actin filaments is modeled as

$$f^{\mathrm{CE}} = f_{\mathrm{o}}^{\mathrm{M}} a \mathbf{f}^{\mathrm{L}}(\ell^{\mathrm{N1}}, \ell^{\mathrm{M}}) \mathbf{f}^{\mathrm{V}}(v^{\mathrm{M}} - v^{\mathrm{N1}}) \tag{2}$$

being proportional to the activation a of the muscle and scaled by the number of available attachment sites $\mathbf{f}^{\mathrm{L}}$ and a modified force-velocity curve $\mathbf{f}^{\mathrm{V}}$. The force-velocity curve $\mathbf{f}^{\mathrm{V}}$ follows Hill's hyperbola for concentric contractions, with a linear extrapolation on the eccentric side: when stretched this modified model will cause the myosin and actin filaments to move together, stretching titin. Since the actin and myosin elements are now independent we have derived an active-force surface $\mathbf{f}^{\mathrm{L}}$ which evaluates how many active sites for cross-bridge attachment are available given the actin-myosin overlap and interference. Finally, the acceleration of the titin and myosin elements are given by

$$\dot{v}^{\mathrm{M}} = \frac{1}{m^{\mathrm{M}}}(f^{\mathrm{T}}\cos\alpha - f^{\mathrm{CE}} + \mathbf{f}^{\mathrm{Z}}(\ell^{\mathrm{N1}}, \ell^{\mathrm{M}})) \tag{3}$$

$$\dot{v}^{\mathrm{N1}} = \frac{1}{m^{\mathrm{N1}}}(f^{\mathrm{CE}} - f^{\mathrm{N1}} + f^{\mathrm{N2}}) \tag{4}$$

where $\mathbf{f}^{\mathrm{Z}}$ is the force developed when two neighboring myosin filaments come into contact (pinching the Z-line), and $f^{\mathrm{T}}\cos\alpha$ is the force of the tendon along the fiber. We use conventional models of activation dynamics, tendon elasticity, and pennation [2] for this model. To test this model we reproduce, in simulation, the ramp lengthening experiments of [6] using the proposed two-element fiber model with titin as a series viscoelastic element.

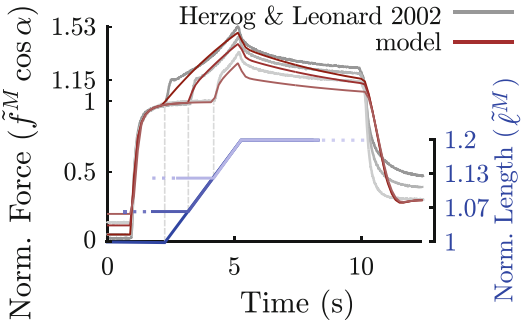

Fig. 3. The simulation results of a maximum-activation ramp-stretch test compared to experimental data [6].

3 Results and Conclusion

The simulation results (Fig. 3) of the ramp-stretch experiments of [6] show that the model is a good candidate: it has a similar short-range and long-range force profile as biological muscle. If titin does function as a series element this will change the way that muscle force is understood to develop, affecting many researchers who study musculoskeletal systems. However, much work remains to be done: these ideas need to be experimentally tested, and many details need to be developed before this model is complete.

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