Simple and Two-Element Hill-Type Muscle Models Cannot Replicate Realistic Muscle Stiffness

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1. Introduction

An important functional property of muscle is to provide stiffness for the limbs [1]. Joint and limb endpoint stiffnesses are critical to control limb posture, movement and interaction with the environment [1,2]. In general, stiffness produces instantaneous resistance to change in muscle length. Stiffness is known to be modulated muscle length (i.e., by joint angles) and muscle activation levels (i.e. α drive) [3], but the mechanisms that produce them remain unclear.

Hill-type models are a class of normalized lumped-parameter models of muscle of varying complexities that can be scaled to approximate specific muscles. They estimate muscle force as functions of muscle architecture (physiological cross sectional area and pennation angle), kinematic state of muscle (length, and velocity) and the muscle activation level (α drive) [4,5]. The goal of this project is to assess the ability of Hill-type models to produce muscle stiffness [6].

2. Muscle Models

2-1. Simple Hill-type model

$$\dot{T}(t) = \frac{K_{SE}}{b} \left(K_{PE} \Delta x + A \right) - \frac{K_{SE}}{b} \left(1 + \frac{K_{PE}}{K_{SE}} \right) T(t)$$

2-2. Two-element Hill-type model

$$\frac{d}{dt}(a_1) + \left[\frac{1}{\tau_{\text{act}1}}(\beta_1 + \left[1 - \beta_1\right]EMG(t - t_{\text{off}}))\right]a_1(t) = \left(\frac{1}{\tau_{\text{act}1}}\right)EMG(t - t_{\text{off}})$$

$$\frac{d}{dt}(a_2) + \left[\frac{1}{\tau_{\text{act2}}} (\beta_2 + [1 - \beta_2] a_1(t)) \right] a_2(t) = \left(\frac{1}{\tau_{\text{act2}}} \right) a_1(t)$$

$$\frac{d}{dt}(a_3) + \left[\frac{1}{\tau_{\text{act3}}}(\beta_3 + [1 - \beta_3]a_2(t))\right] a_3(t) = c\left(\frac{1}{\tau_{\text{act3}}}\right) a_2(t)$$

 $\hat{F}_{\rm f} = \hat{a}(t)\hat{F}_{\rm a}(l)\hat{F}_{\rm v}(v)$

$$F_{\rm m} = c[\hat{F}_{\rm f} + \hat{F}_{\rm p}(l)]\cos\theta$$

Force-length and force-velocity equations

Force-length:

$$\hat{F}_{a}(l) = \frac{((-878.25(l \times 1.253)^{2} + 2200.4(l \times 1.254) - 1192)}{186.24}$$

$$\hat{F}_{P}(l) = \frac{e^{-1.3 + 3.8(l \times 1.253)}}{186.24}$$

Force-velocity:

$$\hat{F}_{v}(v) = \frac{\left(1 - \frac{v}{v_{o}}\right)}{\left(1 + \frac{v}{v_{o}k}\right)} \text{ for } v \le 0 \qquad \qquad \hat{F}_{v}(v) = 1.5 - 0.5 \frac{\left(1 + \frac{v}{v_{o}}\right)}{\left(1 - \frac{7.56v}{v_{o}k}\right)} \text{ for } v > 0$$

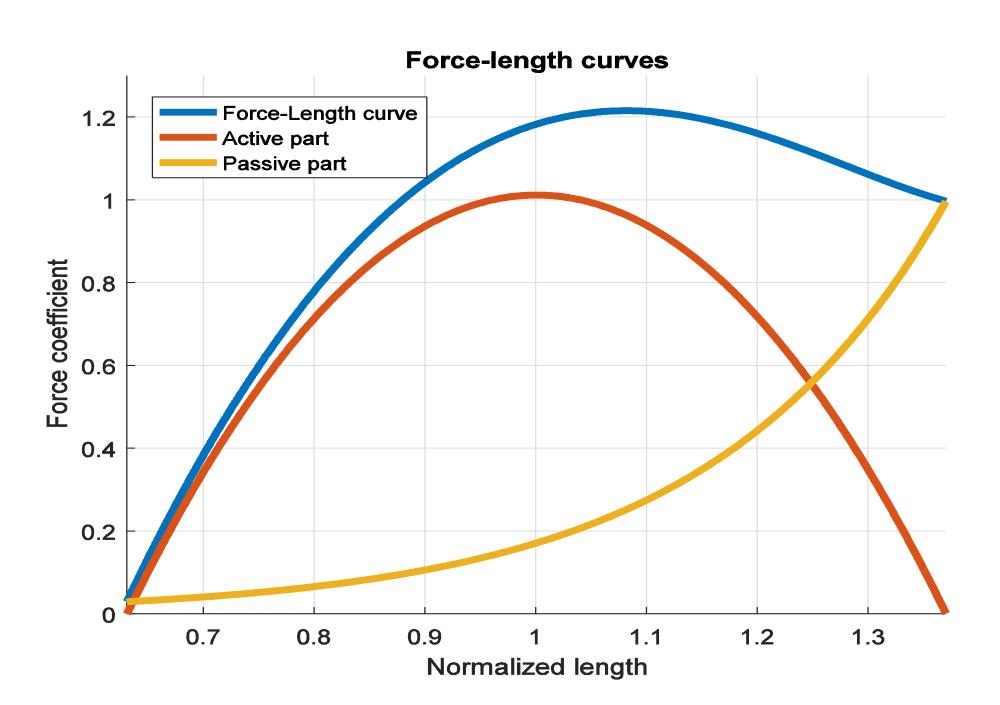


Figure 1: Force-Length curves (active, passive and superposition)

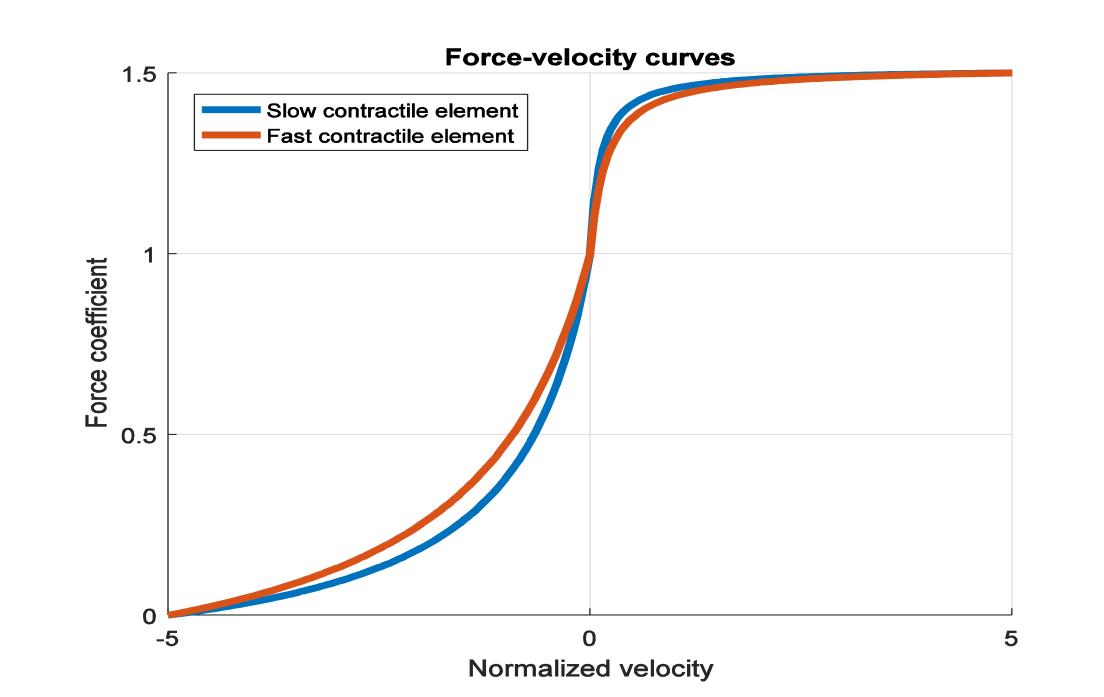


Figure 2: Force-Velocity curves (for slow and fast contractile elements)

3. Methods

Model explanation

In this project, we studied versions of two popular Hill-type muscle models. The first model is a simple linear model consisting of series and parallel springs, a viscous element and a contractile element referred herein as the simple Hill-type model without force-length properties (or the Hill-type w/o fl) [4]. The contractile element converts the α drive to active muscle force. This model, as presented, did not have force-length properties. Thus, we modified it by adding force-length properties to it (i.e., Hill-type w fl). activated. We also calculated values of stiffness at two lengths (0.8 and 1.2 L0) for α drive ranging between 0 to 100 percent in steps of 1%.

The two-element Hill-type model incorporates two parallel active contractile elements for slow and fast muscle fibers (i.e., Two-Element model) [5].

Simulation details

We estimated muscle stiffness in quasi-static condition by applying ten small displacements (of 2.5% L0, where L0 is the optimal muscle length) with the muscle lengths set between 0.5 L0 and 1.8 L0 while the muscle was fully

4. Results and Discussion

Figure 3 shows stiffness for all models as a function of normalized muscle length. To make figures easier to compare, all figures are normalized to their maximum absolute value. Stiffness for the simple Hill-type model without force-length properties does not depend on muscle length (red). Stiffness varies as a function of muscle length for the other two models (blue and green). However, it becomes negative at some lengths. It is clear that the negative stiffness is not physically possible since it results in instability.

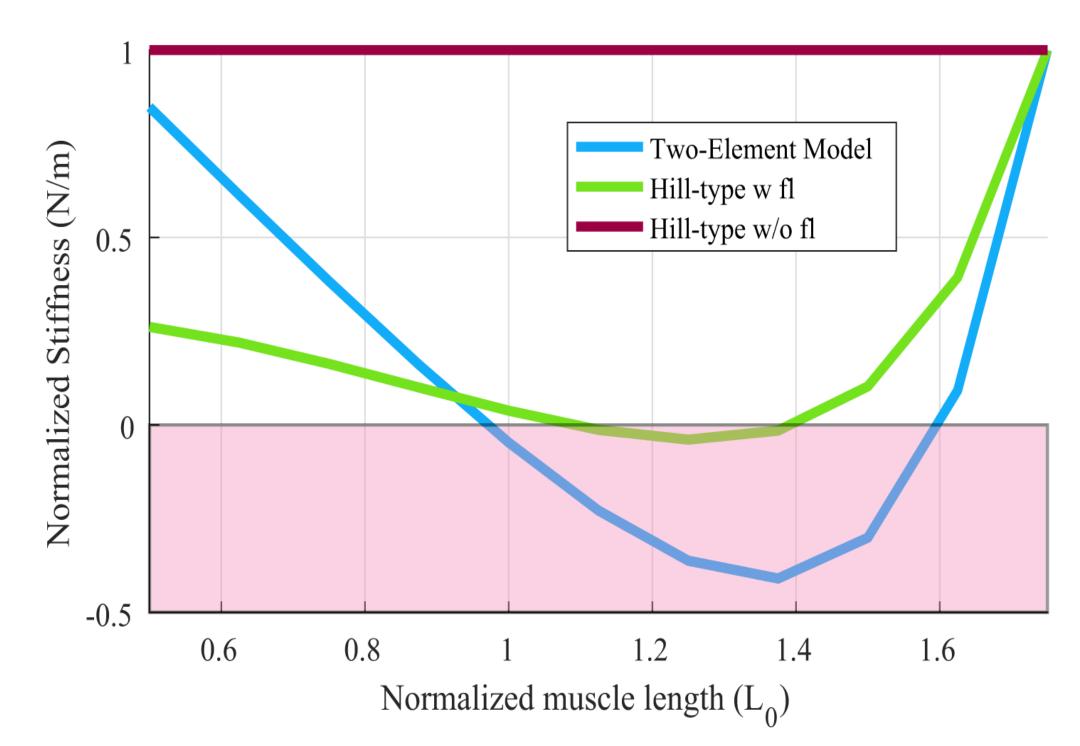


Figure 3: Stiffness as a function of muscle length

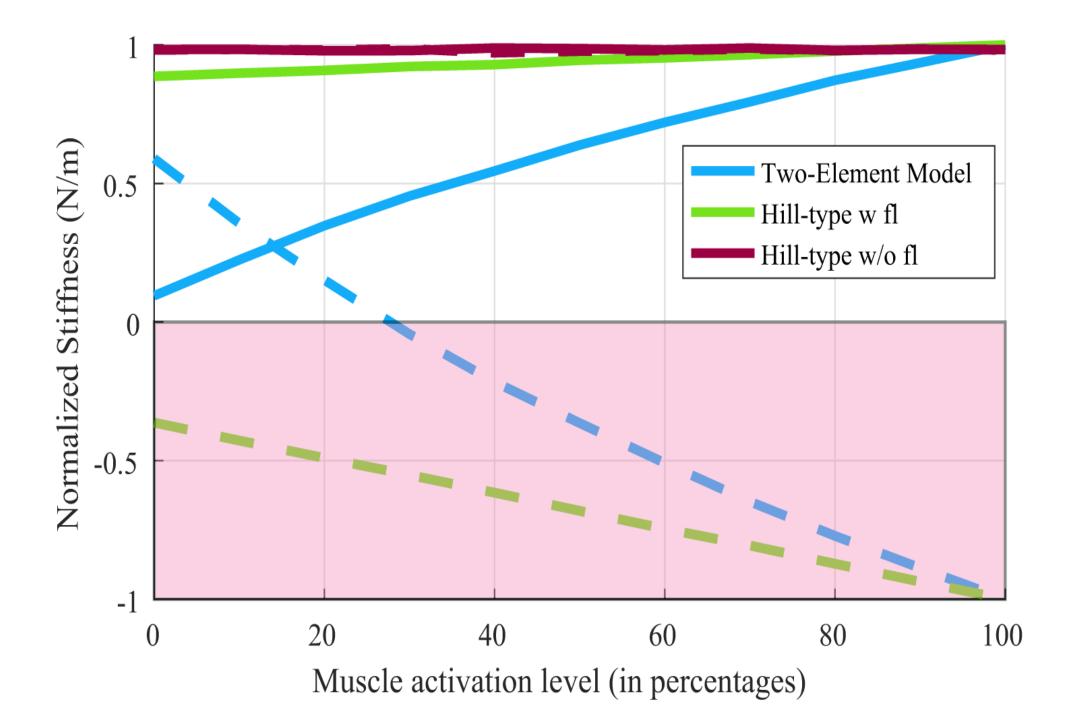


Figure 4: Stiffness as a function of muscle lengths equal to 0.8 L0 (solid lines) and 1.2 L0 (dashed lines).

Figure 4 shows the stiffness for all models as a function of muscle activation level at two representative muscle lengths (0.8, 1.2 L0). Once again, stiffness is not a function of muscle activation in the absence of force-length properties (red). Interestingly, for other two models, change in the stiffness was consistent with the relative proportions of the derivatives of the active and passive parts of the force-length curve. i.e. the more the activation, the larger the weight of the active part. This result is expected considering that activation applies only to the active part of the force-length curve of muscle. As can be seen on the figures, the stiffness can be negative for both length dependent models (blue and green) when the muscle length is longer than L0, which demonstrates that the models fail to replicate realistic muscle stiffness.

5. Discussion

Our results show the simplest Hill-type model fails to reproduce both muscle length and activation dependence of stiffness. The modified and two-element Hill-type muscle models produced stiffness dependence on muscle length and activation, but invariably produce negative stiffness at some muscle lengths, which is not physically realistic. Although force-length properties are very important in explaining stiffness [1,2], Hill-type models cannot replicate realistic muscle stiffness even when including presence of force-length properties.

Future work will explore if dynamic simulations (as opposed to this quasi-static version) and other extensions, such as the inclusion of force-velocity properties, can produce realistic muscle stiffness. If those efforts are unsuccessful, other models such as population-, fiber- and sarcomere-based—although more computationally complex—would need to be preferred.

References:

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