A MUSCLE MODEL FOR INJURY SIMULATION

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Introduction

Car accidents frequently cause neck injuries [1] that are painful, expensive, and difficult to simulate. The movements that lead to neck injury include phases in which the neck muscles are actively lengthened [2]. Actively lengthened muscle can develop large forces that greatly exceed the maximum isometric force ($f_{\rm ISO}$) [3]. Although Hill-type models are often used to simulate human movement [4], this model has no mechanism to develop large tensions during active lengthening. When used to simulate neck injury, a Hill model will underestimate the risk of injury to the muscles but may overestimate the risk of injury to the structures that the muscles protect.

Methods

We have developed a musculotendon model that includes the viscoelasticity of attached crossbridges and has an active titin element (Figure 1). Titin is a giant elastic protein that spans the distance between the Z and M lines. When activated, our model viscously bonds a part of titin to the neighbouring actin filament, effectively halving the length of the segment of titin that is free to stretch. As a result, when the model is activated the stiffness of titin is effectively doubled: the titin-actin bond ensures that only the distal segment is free to lengthen.

To evaluate the model, we simulate the experiments of Leonard et al. [1] that feature extreme active lengthening. The experiment begins by maximally activating the contractile element (CE) at the optimal length. Next the CE is slowly lengthened until the observed force drops. For context, we repeat the simulations using a Hill-type muscle model [4].



Figure 1: The proposed model includes a lumped viscoelastic crossbridge and a two-segment model of titin. When the model is activated the point between two titin segments can form a viscous bond with actin.



Simulation of Leonard, Journaa & Herzog 2010

Figure 2: When an extreme active lengthening experiment [6] is simulated the proposed model develops forces similar to biological muscle.

Results

When the proposed model is activated and lengthened [3], it develops forces that greatly exceed f_{ISO} , similar to biological muscle (Figure 2). As in Leonard et al.'s experiments [5], the model can develop active forces beyond actin-myosin overlap, at least until the titinactin bond slips off the actin filament. In contrast, the Hill-type model's force is far lower.

Discussion

We next plan to evaluate the model by simulating active lengthening experiments that have been done on whole muscle [6] in rabbits. Following this, we will simulate the head and neck movements of a finite element model during an in-vivo whiplash experiment.

References

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Acknowledgements

Financial support by the Deutsche Forschungsgemeinschaft under Germany's Excellence Strategy – EXC 2075 390740016 (SimTech) – is gratefully acknowledged.