MECHANICS OF ECCENTRIC CONTRACTION-INDUCED MUSCLE INJURY

YINGXIN GAO*, ALAN S. WINEMAN*, ANTHONY M. WAAS †, JOHN A. FAULKNER‡

* Department of Mechanical Engineering
University of Michigan
Ann Arbor, MI 48105, USA
lardan@umich.edu

† Department of Aerospace Engineering
University of Michigan
Ann Arbor, MI 48105, USA

‡ Department of Biomedical Engineering
University of Michigan
Ann Arbor, MI 48105, USA

Skeletal muscle is composed of two main components, contractile myofibers and extracellular matrix (ECM). The myofibers adhere to the surrounding ECM through the basal lamina, sarcolemma and two main complexes of adhesion molecules, integrin and dystrophin and dystrophin associated glycoprotein (DAG)[1]. Exercise-induced muscle injury is associated with eccentric (forced lengthening) contraction [2]. Injury somehow involves the mechanical forces during stretch in tearing the tissue. When a muscle is active and stretched, the ECM linking adjacent fibers could break and the basal lamina between the myofiber and ECM could be peeled off the myofiber. Also sarcolemma damage occurs during heavy eccentric exercise. While there are many experimental data showing the structural abnormality following eccentric contraction, found in both animal and human models, there are few mathematical models to study the mechanisms of the injury.

In this paper we will consider a single muscle fiber as a composite system consisting of an extracellular matrix (endomysium) with reinforcement by contractile myofibers, which can generate force and bear loads actively. The sarcolemma, basal lamina and the DAG are coating the myofibers on one side and interface with collagen fibers of the endomysium on its other side [3].

The stress transfer between the contractile myofibers and ECM in a single muscle fiber is studied utilizing the shear lag model, first proposed by Cox [4], and a shear cohesive zone model to simulate damage development in the coating between the myofibers and ECM. It is assumed that the ECM carries only shear stress and the myofibers can only undergo axial contraction or extension. To simulate the eccentric contraction of myofibers, the muscle fiber is stretched to some length and then the myofibers contract, which leads to some interface shear stress between the myofibers and the endomysium. In the region modeled as a cohesive zone, the shear strength is set to τ^* and the mode II fracture energy (this is the energy dissipated through the damaging coating) is set to G_{IIc}. τ^* and G_{IIc} are treated as known material constants. As the force generated in muscle increases, the influence of the damage development on muscle performance is quantified and several results will be presented that illustrate the stress and strain distributions in the various constituents of the single muscle fiber.

References

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