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Finite element analysis of mechanics of lateral transmission of force in single muscle fiber

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ABSTRACT

Most of the myofibers in long muscles of vertebrates terminate within fascicles without reaching either end of the tendon, thus force generated in myofibers has to be transmitted laterally through the extracellular matrix (ECM) to adjacent fibers; which is defined as the lateral transmission of force in skeletal muscles. The goal of this study was to determine the mechanisms of lateral transmission of force between the myofiber and ECM. In this study, a 2D finite element model of single muscle fiber was developed to study the effects of mechanical properties of the endomysium and the tapered ends of myofiber on lateral transmission of force. Results showed that most of the force generated is transmitted near the end of the myofiber through shear to the endomysium, and the force transmitted to the end of the model increases with increased stiffness of ECM. This study also demonstrated that the tapered angle of the myofiber ends can reduce the stress concentration near the myofiber end while laterally transmitting force efficiently.

1. Introduction

Skeletal muscles have a complex hierarchical structure which is mainly composed of myofibers and the extracellular matrix (ECM) surrounding them. There are three different structural levels of the ECM: endomysium that surrounds every single myofiber; perimysium that binds the muscle fascicles; and epimysium that encompasses the entire skeletal muscle. ECM not only provides structural support to ensure integrity of the whole muscle, but more importantly, interactions between the ECM and myofibers determine the mechanical behaviors of skeletal muscles. The unique structural, biochemical and biophysical properties of the ECM make it an important structure in passing mechanical signals into the cell to produce a signaling cascade through molecules that connect the ECM to muscle cells (Lieber, 2002). Changes in the ECM will cause altered mechanical environments around the cell through this interactions, and therefore initiate the mechanotransduction process in muscle (Kjaer, 2004; Purslow, 2002), which affects the muscle adaptation to aging, injury, disease, and the outcomes of corresponding treatments.

The mechanical interactions between the muscle cell and ECM mainly occur through the force transmission between them. It has been shown that for many muscles, across species, muscle fiber commonly ends within the fascicles without reaching the myotendinous junction (Gaunt and Gans, 1992; Trotter, 1993, 2002; Trotter and Purslow, 1992). This suggests that the force generated in these muscle fibers must be transmitted laterally via the endomysium. Such pathway of force transmission was defined as lateral transmission of force (Huijing, 1999; Monti et al., 1999). Although the existence and the necessity of lateral transmission of force have been demonstrated experimentally between single muscle fibers and fascicles, and even different muscles (Balice-Gordon and Thompson, 1988; Huijing et al., 1998; Street and Ramsey, 1965; Street, 1983), the mechanical mechanism of this transmission is not well understood.

Skeletal muscle has a complicated microstructure, and therefore it is difficult to experimentally determine the mechanisms of lateral transmission of force. Mathematical models have the advantages in manipulating variables which is otherwise difficult to do experimentally. The most commonly used mathematical models to describe forces generated in myofibers are the Hill model and the Huxley model. The Hill (1938) model is a phenomenological model in which microstructures of muscles are not incorporated; therefore, it is not able to provide structural mechanisms of muscle contractions (Herzog, 2000). In contrast, the Huxley model is a model including structures that affect the force generation, in which force generated in myofibers is described as a function of attachments of cross-bridges (Huxley, 1957). Zahalak further developed the Huxley model by incorporating chemical changes during muscle contractions and introducing a distribution-moment (DM) method to save computing expense (Zahalak, 1981; Zahalak and Ma, 1990). However, the

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Zahalak model is a model of sarcomeres based on the cross-bridge theory, and it is focused only on myofibers without the surrounding endomysium. Therefore it cannot be directly used to study the interactions between myofibers and the ECM.

Most previous mathematical models considered skeletal muscles as one tissue without separating the structures of myofibers and the ECM, therefore, no information about force transmission between them can be determined from those models (Blemker et al., 2005; Chen and Zeltzer, 1992; Johansson et al., 2000; Kojic et al., 1998; Van der Helm, 1994). Yucesoy et al., (2002, 2003) developed a FE model, in which two single layers of mesh, the muscle fiber layer and the ECM layer, were linked elastically. However, the geometry of that model could not represent the physiological structure of long muscles, in which the muscle fibers terminate intrafascicularly.

The objective of this study was to determine the mechanisms of lateral transmission of force between myofibers and the ECM. We developed a 2D FE model of single muscle fiber with two separate tissues, myofiber and endomysium. The Zahalak model was incorporated in the FE model to describe the active force generated in the myofiber during contractions. Stress distributions along the myofiber and the interface between myofiber and endomysium were analyzed. Parametric studies were performed to determine effects of mechanical properties of the endomysium and tapered end of myofiber on the lateral transmission of force between the myofiber and endomysium.

2. Methods and models

A fiber-reinforced composite is a material that is composed of two different components with discontinuous and strong fibers being embedded in a relatively compliant matrix. Muscle is functionally a fiber-reinforced composite consisting of an ECM with reinforcement by myofibers (Huijing, 1999). The basic structural unit of the muscle is composed of one myofiber and the surrounding endomysium. In this study, the force transmission between the myofiber and the endomysium within such one structural unit, i.e., one single muscle fiber was studied.

2.1. Model description

The geometry of a simplified single muscle fiber, or one structural unit, is shown in Fig. 1, in which the myofiber is surrounded by the endomysium. The interface between the myofiber and the endomysium was modeled as perfectly bonded. For 2D problems, both myofiber and endomysium were modeled to be of rectangular shape. Both ends of the single fiber model were fixed to simulate isometric contraction, by which no injury will be induced. To reduce computational time, only the upper right quarter of the model, which is in the dashed line in Fig. 1, was calculated due to the geometric symmetry of this model. Symmetric boundary conditions were applied on symmetry axes.

Physiological structures of the tapered end of single muscle fiber have been well observed in previous studies (Barrett, 1962; Eldred et al., 1993; Gaunt and Gans, 1990; Trotter, 1990). For the purpose of geometric simplification, a trapezoid shape and a constant angle of fiber end were chosen for tapered ends. Based on the previous observation on the ratio of cross-section area to the taper length (Eldred et al., 1993), effects of tapered end on force transmission were determined by changing the rectangular ends to the ends with 5° and 15° tapered angles (Fig. 2). Although the angles may not reflect the physiological geometry, the purpose of this study is to determine the sensitivity of lateral transmission of force between the myofiber and endomysium to the taper angle.

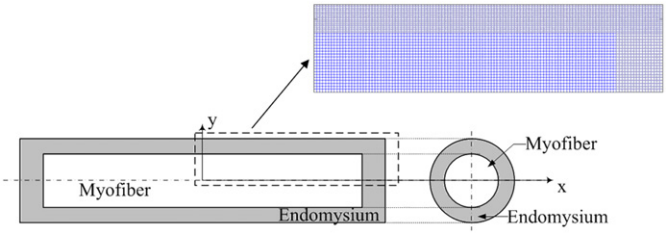

Fig. 1. Schematic diagram of 2D single muscle fiber model. The single muscle fiber is composed of myofiber and the surrounding endomysium.

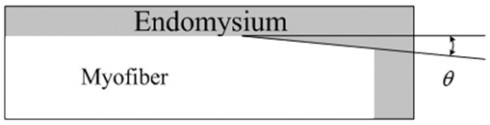

Fig. 2. End of myofiber with $\theta=5^o$ and $\theta=15^o$ tapered angels.

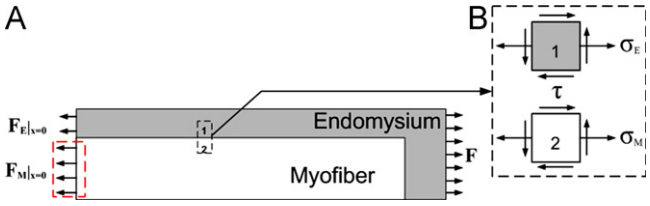

Fig. 3. (A). Free body diagram of the single muscle cell, or the structural unit, during muscle contraction. F is the total force transmitted to the end of the single fiber and is calculated as reaction force at the right end of the single muscle fiber. F_M represents the tensile load in the myofiber, and $F_M|_{x=0}$ represents the force in myofiber at $x=0$. F_E represents the tensile load in the endomysium, and $F_M|_{x=0}$ represents the force in the endomysium at $x=0$. Equilibrium in force requires that $F_M+F_E=F$ at any transversal cross section along x. The force in myofiber is $F_M=F_a+F_p$, in which F_a and F_p are the active force and the passive force in the myofiber, respectively. (B). Stress state of elements in the endomysium and the myofiber.

A 2 s, 100 Hz stimulation signal was applied to myofibers to induce a tetanic contraction. Total force transmitted was calculated as the reaction force at the right end of single muscle fiber, and stress distributions of the interfacial shear stress, τ, and the tensile stress in the myofiber, σ were calculated (Fig. 3). The role of ECM on force transmission was then determined by changing the stiffness of the endomysium.

2.2. Modeling active stress of myofibers

Active stress in myofiber during contraction is calculated using Zahalak's model (Zahalak and Ma, 1990) in which distribution of bonded cross-bridge density $n(x,t)$ is determined by net attachment and detachment rates of bonded cross-bridge, $f(x)$ and $g(x)$, and the relative shortening velocity $v(t)$ between actin and myosin:

$$\frac{\partial n}{\partial t}-v(t)\frac{\partial n}{\partial x}=r([Ca])f(x)(1-n)-g(x)n \tag{1}$$

where $r([Ca])$ is a function of sarcoplasmic free calcium concentration defined as

$$r([Ca])=\frac{k_1^2[Ca]^2}{k_1^2[Ca]^2+k_1k_{-1}[Ca]+k_{-1}^2} \tag{2}$$

In Eq. (2), [Ca] is the concentration of free Ca^{2+} ions in the sarcoplasm; k_1 and k_{-1} are binding and release rates of calcium, respectively, which can be determined by stimulation signals and initial calcium concentrations. The distribution-moment method developed by Zahalak, (1981) is then used to calculate force generation. For a more detailed method for calculating the active stress in the myofibers, please refer to Appendix A or previous study by Zahalak, (1981).

2.3. FEM implementation of the model

To reduce the complexity of muscle contraction, the contraction process was modeled as small discretized time steps in this study. Within each step, the muscle contraction was modeled as a quasi-static problem, and viscoelastic behaviors of the model were neglected. The principle of virtual work was applied to solve for nodal displacements at each time step. The Total Lagrange (TL) formulation, in which all quantities are measured with respect to the original configuration at t_0, is used to describe the deformation. The principle of virtual work at the time $t+\Delta t$ is described as

$$\int_{{}_0V} {}^{t+\Delta t}_{0}S_{ij}\,\delta\,{}^{t+\Delta t}_{0}E_{ij}\,d^0V={}^{t+\Delta t}\mathbf{R} \tag{3}$$

where:

$${}^{t+\Delta t}_{0}S_{ij}={}^{t}_{0}S_{ij}+{}_0S_{ij} \tag{4}$$

$${}^{t+\Delta t}_{0}E_{ij}={}^{t}_{0}E_{ij}+{}_0E_{ij} \tag{5}$$

in which ${}_0\mathbf{S}$ and ${}_0\mathbf{E}$ are the incremental growth of the stress and strain tensors, respectively, from time t to time $t+\Delta t$. ${}^{t}_{0}\mathbf{S}$ and ${}^{t}_{0}\mathbf{E}$ are the second PK stress tensor