A MICRO-STRUCTURAL MUSCLE FIBRE MODEL SHOWS TENSION–COMPRESSION ASYMMETRY MECHANISMS

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INTRODUCTION

Skeletal muscle tissue presents a highly asymmetrical behaviour when passively loaded in tension or compression, but no structural model addressing this has been reported. Passive skeletal muscle derives its structural response from titin filaments, collagen fibres in the connective tissue and incompressibility due to high fluid content. The objective of this paper is to develop a micro-structurally based mathematical model to predict the known tension-compression asymmetry. Such a model is a precursor to future constitutive formulations for finite element modelling.

METHODS

One cylindrical muscle fibre surrounded by connective tissue was considered. The collagenous fibres of the endomysium and perimysium were grouped and modelled as randomly oriented wavy helices wrapped around the muscle, see Figure 1.

Figure 1. Model schematic showing cylindrical muscle fibre wrapped in helical collagenous connective.

The titin filaments are tension only springs. The model calculates the force developed by the titin molecules (F_{titin}) and the collagen network (F_p) when the muscle fibre undergoes a stretch (λ) as follows:

$$F_{res}(\lambda) = \begin{cases} F_z + F_{titin} - F_p & \lambda \geq 1 \\ F_p - F_z & \lambda < 1 \end{cases} \quad (1)$$

$$F_z(\lambda) = \int_{\theta_{min}}^{\theta_{max}} A_C p_H(\theta_0) \left[\frac{\lambda \sin \theta_0}{\lambda_H(\lambda, \theta_0)} \right]^2 \sum_{k=0}^{n_f} \frac{1}{n_f} \sigma_H(E_c, \lambda, \theta_0, w(k)) d\theta_0 \quad (2)$$

$$F_p(\lambda) = \int_{\theta_{min}}^{\theta_{max}} \frac{A_C}{2} p_H(\theta_0) \left[\frac{\cos \theta_0}{\lambda_H(\lambda, \theta_0) \sqrt{\lambda}} \right]^2 \sum_{k=0}^{n_f} \frac{1}{n_f} \sigma_H(E_c, \lambda, \theta_0, w(k)) d\theta_0 \quad (3)$$

$$F_{titin}(\lambda) = A_F n_1 \frac{k_B T}{L_p} \left(\frac{\lambda-1}{n_2 L_c} + \frac{1}{4(1-n_2 L_c)^2} - \frac{1}{4} \right) \quad (4)$$

The model parameters are all physical: the collagen modulus (E_c), proportion of connective tissue (p_c), minimal and maximal waviness (w_{min}, w_{max}), distribution of initial angles of collagen fibres (p_c), parameters of fibre family beta distribution (α, β) titin persistence and contour length (L_p, L_c), number of titin molecules per muscle area (n_1) and number of titin molecules per muscle fibre length (n_2). The term F_p shows the pressure contribution.

The model was evaluated using a range of literature based realistic input parameters and compared to the experimental data available [1-5].

RESULTS

Figure 2 shows the model response corridor over the range of input parameters applied compared to the range of responses found in the literature [1-5] for which only extrema curves are shown for clarity.

Figure 2. Model response range and the corresponding experimental data from the literature.

DISCUSSION

The non-linearity and tension compression asymmetry were well captured by this structural model. The titin filaments are predominant at low tensile stretch ratios, but for higher stretches the collagen network was responsible for most of the stiffness. The initially wavy angled collagen fibres account for the non-linear response since, as the collagen fibres are being recruited, they straighten and re-orient. The asymmetry of compression/tension response was found to result from the pressure contribution in compression. Although the model presented considers only a single muscle fibre, this is the first model to provide a micro-structural explanation for the tension-compression asymmetry observed in muscle tissue. For along-fibre tension and compression respectively, the collagen network contributes to tissue stiffness through two different mechanisms respectively: 1) a longitudinal force directly opposing tension and 2) a pressure on incompressible muscle fibres opposing compression.

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