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Stretch-Twist coupling in collagen leads to hysteresis

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Background

The extracellular matrix (ECM) of cardiac tissue plays a critical role in the observed mechanical function of the heart. Pathologies such as ventricular hypertrophy and congestive heart failure involve significant remodelling of the ECM. Despite advances in experimental technology enabling detailed measurement, mathematical models that describe the nature of interactions between the ECM and cardiomyocytes are still lacking.

Here we explore the role of collagen during loading and unloading of passive ventricular muscle tissue. The mechanical behaviour shows hysteresis, Fig. 1.

Contribution

While most studies claim that the hysteresis is due to the dissipative viscous components of the tissue. They do not provide a direct association to the components that give rise to the behaviour. Here we show that the perimysial collagen can exhibit two mechanical state that give rise to hysteresis.

Proposed mechanism

The cross-linking topology of the ECM is crucial to the observed elastic behaviour of the ventricular tissue.

The mechanics arise from the ability of the cardiomyocytes (white cylinder) to deform while being embedded in the matrix (grey domain), Fig 2. Cardiomyocytes remain highly deformable, but only as far as their cross-linking constraints allow.

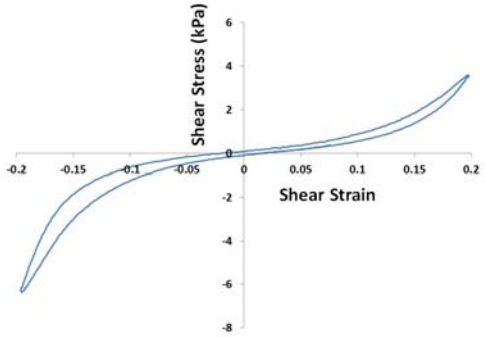

Fig 1. Ventricular muscle tissue mechanics

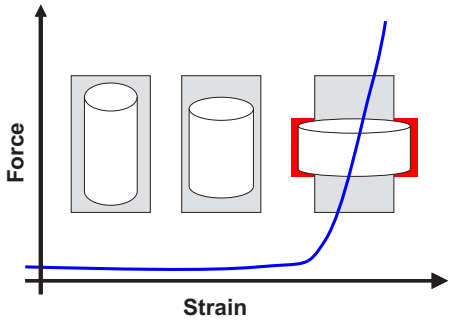

Fig 2. Proposed Mechanism for Soft elasticity

Hypothesis

The difference in the mechanical behaviour occurs when the embedded matrix deforms differently while unloading than while loading. We test this hypothesis by investigating the mechanical behaviour of the key components that compose the matrix.

Model of perimysial collagen

Following [1] we assume the perimysial collagen fibre to be arranged as a helical spring with a uniform circular cross-section. Such elastic filaments can been modelled as Kirchhoff rods. The expression for free energy in terms of the angular strain vector can be obtained using Kirchhoff's theory:

$$E = \frac{A_1}{2}\int_0^L ds\,(\Omega_1)^2 + \frac{A_2}{2}\int_0^L ds\,(\Omega_2-\kappa_0)^2 + \frac{C}{2}\int_0^L ds\,(\Omega_3-\tau_0)^2$$

Where A_1, A_2 are the bending rigidities and C is the torsional rigidity. Using the microstructural parameters for rat perimysial collagen reported in [1], we constructed a discrete model of a perimysial collagen fibre.

Virtual loading/unloading protocol

The fibre model was quasi-statically loaded by homogeneously stretching it along the axis and then homogeneously relaxing it in uniform steps. Specifically, at each step, the fibre is deformed along its axis and the configuration for minimum E is determined.

Results

The force $\mathrm{F} = {}^{\partial E}/_{\partial x}$ vs strain profile of the perimysial collagen model under loading and unloading conditions are shown in Fig. 3. The profiles show the existence of a bistable equilibrium in the conformation space of the model elastic filament. The filament shape along these paths are different (Fig 3. insets), this indicates that the filament may operate under two states-- coiled and stretched state [2].

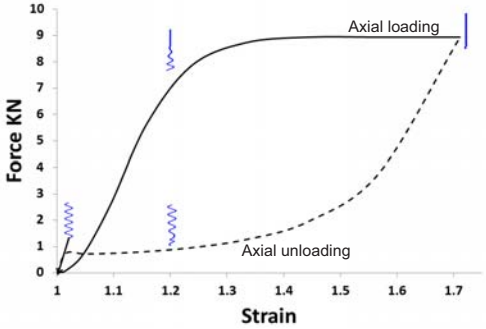

Fig 3. Perimysial collagen mechanics

Summary

The force generated by perimysial collagen fibre in the coiled state is much larger for the same fibre in stretched state.

The engagement of the torsional degree of freedom in the mechanics lies at the genesis of these two states.

For a very small *C* the engagement of the torsional degree of freedom is limited and the filament behaves like a neo-Hookean spring.

Torsional rigidity depends on local hydrodynamics. Changes in MMP's and interstitial fluid content, modify C and therefore collagen and muscle mechanics.

Future work

Develop a multiscale model, for ventricular muscle tissue mechanics, that explicitly represents the structure and mechanics of the collagen fibres.

References

[1] Mackenna et al, Am. J. Physiol., 273:H1576-1586

[2] P. G. de Gennes, J. Chem. Phys., 60:5030-5042