Towards Patient Specific Modeling of Soft Biological Tissues

Mikhail Itskov
Department of Continuum Mechanics, RWTH Aachen University, Germany
itskov@km.rwth-aachen.de

Kevin Linka
Department of Continuum Mechanics, RWTH Aachen University, Germany
linka@km.rwth-aachen.de

Vu Ngoc Khiêm
Department of Continuum Mechanics, RWTH Aachen University, Germany
vu@km.rwth-aachen.de

Markus Hillgärtner
Department of Continuum Mechanics, RWTH Aachen University, Germany
hillgaertner@km.rwth-aachen.de

Abstract

The mechanical response of human soft tissues strongly varies between individuals and depends on many factors like for example age, sex, health state etc. The differences in the stress can then reach 300% or even more (see, e.g. [1]). Against this background, application of phenomenological models to patient specific simulations appears to be meaningless. Indeed, such models can provide realistic predictions only on the basis of experimental data which require invasive procedures for harvesting of test samples. Under these circumstances, it is more promising to rely the modeling on the features common for all soft tissues. These are, first of all, the micro-structure of soft tissues and their chemical composition. In this context, collagen is the most important component which dominates the mechanical response of soft fibrous tissues. Collagen fibers represent an assembly of collagen fibrils embedded into a proteoglycan rich matrix. In turn, the collagen fibrils are build from tropocollagen molecules. This hierarchical structure is just common for all soft tissues. The difference in their response is only due to various amounts collagen, orientational distributions of fibers, dimension and packing density of collagen fibrils as well as extended fiber lengths of the tropocollagen molecules. All these data can be obtained for a particular tissue by a minimal invasive procedure and used to inform a multi-scale model of collagen.

Such a model is proposed in the present contribution (see also [2, 3]). Accordingly, loading of the tissue triggers sliding between fibrils and adjacent proteoglycan bridges [4]. The tropocollagen molecules inside a fibril can be in an entropic or energetic state depending solely on their

current configuration. A statistical framework is utilized in order to describe a physically motivated transition between these states of tropocollagen molecules. In addition, deformations of a single tropocollagen molecule are affected by intensity of the cross-links to adjacent molecules. Furthermore, the damage at the molecular scale is elucidated by the breakage of intramolecular hydrogen bonds [5]. The so obtained model demonstrates good agreement to various experimental data available in literature and could serve in future as a basis for the patient specific tissue simulations mentioned above.

References

[1] G. A. Holzapfel, G. Sommer, Ch. T. Gasser and P. Regitnig, Determination of layer-specific mechanical properties of human coronary arteries with nonatherosclerotic intimal thickening and related constitutive modeling, *American Journal of Physiology - Heart and Circulatory Physiology*. 289(5):H2048–H2058, 2005.

[2] K. Linka and M. Itskov, Mechanics of collagen fibrils: a two-scale discrete damage model, *Journal of the Mechanical Behavior of Biomedical Materials*, 58:163–172, 2016.

[3] K. Linka, V. N. Khiêm and M. Itskov, Multi-scale modeling of soft fibrous tissues based on proteoglycanmechanics, *Journal of Biomechanics*, 49:2349–2357, 2016.

[4] S. Rigozzi, R. Müller and J. G. Snedeker, Local strain measurement reveals a varied regional dependence of tensile tendon mechanics on glycosaminoglycan content, *Journal of Biomechanics*, 42(10):1547–1552, 2009.

[5] S. P. Veres and J. M. Lee, Designed to Fail: A Novel Mode of Collagen Fibril Disruption and Its Relevance to Tissue Toughness, *Biophysical Journal*, 102:2876–2884, 2012.