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Living tissues are naturally prestrained (or, equivalently, residually stressed), as clearly illustrated by Y.C. Fung's famous opening angle experiment on arteries [1]. If the biomechanical role of residual stresses has been well described for many tissues, their origin remains an open question; growth, however, is a likely candidate. Indeed, differential (i.e., heterogeneous) growth induces residual stresses, in order to accommodate for the non-compatible growth [2]. In continuum models of growth, growth-induced residual stresses have been either kept [2] or dismissed [3], depending on the modeling objective: growth-induced residual stresses allow to quantitatively reproduce the opening angle experiments on the left ventricle [2], on the other hand they can lead to inconsistencies in the mechanical response of hypertrophied ventricles [3]. Moreover, only a partial organ growth is required to induce residual stresses that are compatible with measured opening angle [2], suggesting that not all residual stresses induced by the complete growth process are present in the mature organ.

In this work I propose a new model of soft tissue biomechanics, including both strain- or stress-driven growth as well as relaxation of residual stresses. It is written in the general framework of finite deformation continuum mechanics, and based on the multiplicative decomposition of the deformation gradient as illustrated on the figure: the reference configuration, denoted Ω_0 , with prestrain field F_0 , deforms into the current configuration Ω through mapping Φ with gradient $F = \nabla \Phi$; now the total transformation is composed into growth, prestrain, relaxation and loading parts such that the elastic deformation gradient is $F_e = F F^{-1} F_r F_p F_0$. In addition to the constitutive relation that relates this elastic deformation gradient to stress, two additional evolution laws are needed to close the model: for growth, and relaxation. Depending on the ratio between the time constants of both laws, the model allows controlling the part of growth-induced residual stresses that is kept in the tissue vs. the part that is relaxed away.

From the computational perspective, in order to compute all terms in F_e , two computations must be performed in parallel: the deformed configuration Ω , obviously, but also the unloaded configuration Ω_r .

In this presentation, I will describe the new modeling framework as well as its implementation in a finite element solver, and multiples illustrations to living tissues.

[1] Chuong and Fung, "Residual Stress in Arteries", Frontiers in Biomechanics, 1986.

[2] Genet et al., "Heterogeneous Growth-Induced Prestrain in the Heart", Journal of Biomechanics, 2015.

[3] Lee et al., "An Integrated Electromechanical-Growth Heart Model for Simulating Cardiac Therapies", Biomechanics and Modeling in Mechanobiology, 2015.

