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A poroelastic model for tissue growth with stress-dependent proliferation

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Abstract

The mechanical environment plays a fundamental role in tissue development, tumor progression, and tissue engineering applications. Understanding how growing tissues interact with their surroundings requires mathematical models that capture the complex interplay between solid mechanics, fluid transport, nutrient diffusion, and cellular response. In this work, we present a biphasic poroelastic framework for modeling tissue growth that integrates these coupled phenomena. We model the tissue as a poroelastic medium. The solid phase comprises the extracellular matrix and embedded cells, and the fluid phase represents the interstitial fluid that permeates the tissue. Growth-induced residual stresses have been experimentally observed to inhibit cell proliferation. We incorporate this mechanobiological feedback through a stress-dependent growth law where the local proliferation rate decreases with increasing compressive stress, in agreement with experimental observations in multicellular spheroids and solid tumors. Furthermore, nutrient transport through the interstitial fluid modulates cellular proliferation, creating a feedback loop that couples tissue growth with mechanical stress and fluid flow. For the numerical simulation of the proposed model, we adopt a mixed formulation of the coupled multiphysics problem. The poroelastic system is discretized with piecewise quadratic elements for displacements, lowest order Raviart-Thomas elements for the fluid flux, and piecewise constant elements for the pressure (P2-RT0-P0), which ensures inf-sup stability. The nutrient

transport equation is discretized using the lowest order Raviart-Thomas elements for the flux and piecewise constant elements for the concentration, which provides local mass conservation and naturally incorporates the coupling with the fluid velocity field computed from the poroelastic problem. Within each time step, the resulting nonlinear system is solved using a splitting strategy, decoupling the poroelasticity system from the mass conservation equations (cellular density and nutrients) and using fixed-point iterations until convergence in each subsystem. We present numerical simulations demonstrating the development of necrotic cores in nutrient limited conditions and the approach to homeostatic states where tissue growth and death achieve a dynamic equilibrium.

Keywords:

poroelasticity tissue growth mechano-chemical regulation