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Mechanical control and reversal of cell fate in living tissues

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Abstract

Tissue mechanics drives influence cell behavior, and cell behavior drives tissue mechanics. The cellular pathways for this recursive feedback are so redundant that pharmaceutical treatment for pathologies of such feedback have limited efficacy. A key example of this is pulmonary fibrosis. This talk will present our integrated experimental and computational work demonstrating that ECM mechanics can serve as a therapeutic target. We show that stress anisotropy drives the fibroblast-to-myofibroblast underlying fibrosis, a transition accelerated through a self-reinforcing feedback loop and long-range mechanical communication through the ECM. This communication is governed by ECM crosslinking. Findings revealed architecture-dependent mechanical responses: physiological dynamic stretching disrupts advanced glycation end-product crosslinks in porous lung-like scaffolds but not in fibrous liver-like matrices. This structure-property relationship directly modulates the nonlinearity of networked solids by controlling mechanical crosslinking. Leveraging these insights, we developed a non-invasive protocol that reverses pulmonary fibrosis in mice by physically disrupting pathological ECM crosslinks.

Keywords:

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