

# Active Fluid-Solid Interaction in Tissue Invasion: Decoupling Indentation and Degradation Mechanics

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## Abstract

The penetration of active cellular tissues into the fibrous extracellular matrix (ECM) is a critical mechanical process driving biological phenomena ranging from embryonic morphogenesis to cancer metastasis. While the resulting engulfment morphologies often appear identical, they originate from fundamentally distinct driving mechanisms: mechanical indentation induced by active cellular tensions (elastocapillarity) versus biochemical degradation of the substrate. Disentangling these modes is crucial, as the mechanical state of the ECM dictates cellular fate through mechanotransduction.

In this work, we present a rigorous continuum mechanical framework to investigate the competitive dynamics between these two modes. We model the cellular aggregate as a viscous active fluid droplet interacting with a hyperelastic solid substrate. The problem is formulated using a Nitsche-Onsager variational approach [1], which naturally handles the energy dissipation and force balance at the fluid-solid interface. To address the computational challenges inherent in multi-phase flows with non-material moving boundaries, we employ a numerical framework utilizing non-conforming meshes and a phase-field description.

We demonstrate the high predictive robustness of this approach through validation against analytical wetting theories and experimental data on elastocapillarity [3] and human colon carcinoma (HCT-8) cell colonies [2]. Leveraging this framework, we analyze the mechanical equilibrium of engulfing tissues, revealing a fundamental mechanosensing inhibition mechanism: substrate tension acts to negatively regulate the degradation rate. Furthermore, our relaxation simulations indicate that this interaction leaves a permanent *invasive signature*, a residual deformation field that persists in the substrate even after tissue removal. This finding suggests that the history of the competition between active stress and matrix remodeling is mechanically encoded in the ECM.

## References

- [1] G. Cicconofri, P. Blanco, G. Vilanova, Sáez P., and Arroyo M. Active interfacial degradation/deposition of an elastic matrix by a fluid inclusion: Theory and pattern formation. *Journal of the Mechanics and Physics of Solids*, 191:105773, 2024.
- [2] X. Shi, Z. Liu, L. Feng, T. Zhao, C.-Y. Hui, and S. Zhang. Elastocapillarity at cell-matrix contacts. *Physical Review X*, 12:021053, 2022.
- [3] R. W. Style, R. Boltyanskiy, Y. Che, J. S. Wettlaufer, L. A. Wilen, and E. R. Dufresne. Universal deformation of soft substrates near a contact line and the direct measurement of solid surface stresses. *Phys. Rev. Lett.*, 110:066103, 2013.