

Abstract Submitted  
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**Modeling mechanical interactions between cancerous mammary acini** JEFFREY WANG, Harvard University, JAN LIPHARDT, Stanford University, CHRIS RYCROFT, Harvard University — The rules and mechanical forces governing cell motility and interactions with the extracellular matrix of a tissue are often critical for understanding the mechanisms by which breast cancer is able to spread through the breast tissue and eventually metastasize. *Ex vivo* experimentation has demonstrated the the formation of long collagen fibers through collagen gels between the cancerous mammary acini responsible for milk production, providing a fiber scaffolding along which cancer cells can disorganize. We present a minimal mechanical model that serves as a potential explanation for the formation of these collagen fibers and the resultant motion. Our working hypothesis is that cancerous cells induce this fiber formation by pulling on the gel and taking advantage of the specific mechanical properties of collagen. To model this system, we employ a new Eulerian, fixed grid simulation method to model the collagen as a nonlinear viscoelastic material subject to various forces coupled with a multi-agent model to describe individual cancer cells. We find that these phenomena can be explained two simple ideas: cells pull collagen radially inwards and move towards the tension gradient of the collagen gel, while being exposed to standard adhesive and collision forces.

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