Title of the dataset

Videos supporting the paper: 2D morpho stability

Files and descriptions

The files in this online resource correspond to figures 4 and 6 in the paper *"Stability of a two-dimensional morphoelastic model for post-burn contraction"*. Within this paper, we consider a two-dimensional morphoelastic model that predicts contraction in scars. This paper is a follow-up for our previous stability analysis in 1D. We study the model's stability. The following videos show the evolution of the effective strains and chemicals in the case of a low, but not too low, signaling molecule decay rate (which yields linear instability for the model). The following descriptive text can also be found in the paper.

Files corresponding to Figure 4. For the effective strains ε_{11} and ε_{22}, the tensions in the densities are diagonal and peak on day 5. The peaks diminish in magnitude in the first 51 days. The effective strain ε_{12} density shows the same intensity of variations on day 5, which are circular and alternating between positive and negative values, like in the stable simulation. However, this pattern changes after 51 days. Unlike the equilibria found in the simulation with stable parameter values, the effective strain densities increase intensely in variation up to day 223. For the effective strain ε_{11} this is an increase on the right edge of the computational domain, for the effective strain ε_{22} on the top edge, and the effective strain ε_{12} around the top right corner. In opposite directions, these densities decreased. For example, we see a decrease on the vertical axis of symmetry for the effective strain ε_{11}. After these peaks of intensities in the effective strain densities on day 223, the densities gradually decrease until day 351 and increase in intensity until they reach equilibrium on day 1200. The effective strain densities oscillate around the (new) equilibria. Compared to the simulation with stable parameter values, we see an increase in the intensity of the same order for $\varepsilon_{12,}$ albeit with more significant numbers. We note that the order of magnitude may also result from the larger wavenumber (k=2) in the initial perturbations.

Files corresponding to figure 6. The early evolution of the chemicals for a low signaling molecule decay rate is comparable to the evolution of the chemicals for stable values. In the first 51 days, the perturbed fibroblast and myofibroblast cell densities move gradually toward equilibria, and the perturbed collagen density moves gradually toward equilibrium in the first 119 days. However, the perturbed signaling molecule density does not move to the expected equilibrium.

Unlike the perturbed signaling molecule density evolution for stable parameter values, the perturbations in the signaling molecule density do not disappear in the first 13 days for an unstable signaling molecule decay rate. The initial peaks decrease in the first few days. At the same time, these peaks merge and shift toward the origin as they decrease further in the first five days. The peaks continue merging, completed within 13 days; however, the signaling molecule density increases strongly in the origin of the computational domain. In the beginning, this increase does not significantly affect the other chemicals; however, after day 51, it causes a considerable difference.

On day 51, it seems that the fibroblast cell density is in the equilibrium N = 10000 cells/cm^3, the myofibroblast cell density in the equilibrium M = 0 cells/cm^3, the signaling molecule density in the equilibrium c = 0 g/cm^3, and the collagen density is around the equilibrium rho = 0.1125 g/cm^3. However, these densities do not stay in and around equilibria. Note the orders of the signaling molecule concentration: 10^{-10} g/cm^3 on day 200 compared to the order 10^{-14} g/cm^3 on day 13. After day 13, in the origin of the computational domain, the signaling molecule density increases

enormously until day 200, after which the density drops back toward equilibrium until day 317. The signaling molecule density then rises to a new equilibrium on day 1200, which shows a clear oscillation. Since the signaling molecule density increases so much up to day 200 in the origin of the computational domain, the fibroblast cell density decreases because of myofibroblast differentiation, and the collagen density increases there. These changes in densities are because signaling molecules stimulate the differentiation and production of myofibroblasts, stimulate the production of collagen, and inhibit the decay of collagen. Further, myofibroblasts also stimulate collagen production. The myofibroblast cell density reaches a maximum on day 213, the collagen density on day 235, and the fibroblast cell density on day 245. After the signaling molecule density reaches a minimum on day 317, we see that the myofibroblast cell density reaches a minimum on day 337, the collagen density on day 375, and the fibroblast cell density on day 448. After these days, such an oscillating effect around new equilibria is visible, which converges on day 1200. The result is a permanently reduced number of fibroblasts, a permanently increased number of myofibroblasts, and a permanently elevated concentration of signaling molecules and collagen, at the origin of the computational domain (i.e., the center of the burn). Taken together, with an unstable signaling molecule decay rate not too low, the numerical method initially seems to behave like a stable regime. This stable behavior changes at a later stage of simulation time, where the numerical method behaves stable enough to let the chemical densities reach new equilibria in an oscillatory way.

From a biological perspective, we can state that an increased expression of signaling molecules (because of their reduced decay) can lead to a period in which a wound fluctuates in contraction. This contraction fluctuation is because the number of (migrating) myofibroblasts increases and decreases. In the beginning, the wound can heal well. However, because of the continued signaling, the scar will fluctuate in thickness and stiffness because of the present collagen concentration. The scar is also highly subject to contraction because of the abundance of myofibroblasts present. The abundance of myofibroblasts and the increased collagen concentration may signify hypertrophy.

Methodology

We used the finite element method and implemented the equations in Matlab. This way, we were able to produce the videos.

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