

**Title of the dataset:** *Video's supporting the paper: 1D morpho stability*

**Files and descriptions:** The files in this online resource correspond to the figures in the paper "*Stability of a one-dimensional morphoelastic model for post-burn contraction*". Within this paper, we consider a one-dimensional morphoelastic model that predicts the contraction in scars. Contraction is the process in which the surrounding tissue is pulled inwards, both reducing the size of the wound and contracting the tissue, possibly resulting in a contracture that makes daily rituals hard to accomplish. In the paper, we analyse the model's stability. The following videos show the evolution of the components of the model (cells, growth factors, collagen, the velocity with which the tissue displaces, and the effective strain present during the deformation), in cases of (in)stability and real/complex eigenvalues. The following descriptive text can also be found in the paper.

**Files corresponding to Figure 2.** The videos show the evolution of the constituents and the mechanics after perturbations in case stability constraints are met and eigenvalues are real-valued. Parameter values are  $\delta_t = 5 \cdot 10^{-4}$  and  $\mu = 100$ . We used a wave with  $k = 1$ .

Fig2 c.avi: the signaling molecules. Because of the boundary condition, the signaling molecule density is fixed at equilibrium on the left boundary of the domain of computation. We see that on the right boundary, the density increases in the first days, after which it decreases to the equilibrium  $c = 0$ .

Fig2 M.avi: the myofibroblasts. The distribution moves to the left, and moves gradually toward the equilibrium  $M = 0$ . Only the values on the right boundary move away from the equilibrium in the first 10 days, because of the differentiated fibroblasts.

Fig2 N.avi: the fibroblasts. During the simulation, the fibroblast distribution displacements to the left, and values above the equilibrium gradually move toward the equilibrium  $N = 10^4$ . The fibroblast distribution on the right boundary starts by moving away from the equilibrium as the fibroblasts differentiate to myofibroblasts because of the increased density of signaling molecules. After the signaling molecule density is almost zero around the right boundary on day 30, the fibroblast distribution moves toward the equilibrium, reaching it fully around day 400.

Fig2 rho.avi: collagen. The plot of collagen is like the plot of the effective strain, although the effect of the local displacements seems larger for collagen, and for collagen it takes much longer before the density reaches the equilibrium  $\rho = 0.1125$ .

Fig2 v.avi: the displacement velocity. Because of the negative values of the displacement velocity density after 12 hours, the mesh moves to the left.

Fig2 e.avi: the effective strain. We see that the displacement velocity density rearranges to negative values. As the density moves below zero, the amplitude of the wave initially increases, after which the density moves gradually toward the equilibrium  $v = 0$ . The effective strain density does not change signs. The values on the boundaries of the domain of computation initially move away from the equilibrium, where all other values gradually move toward the equilibrium  $\varepsilon \approx -0.05$ .

Overall, the model behaves absolutely stable given these stable parameter values.

**Files corresponding to Figure 3.** The videos show the evolution of the constituents and the mechanics after perturbations in case the stability constraints are met, but the mechanical eigenvalues are complex-valued. Parameter values are  $\delta_c = 5 \cdot 10^{-4}$  and  $\mu = 1$ . Initially, the time step is  $\delta_t = 0.01$ , which changes to  $\delta_t = 1$  after 2 days and to  $\delta_t = 2$  after 50 days. We used a wave with  $k = 1$ .

Fig3 c.avi: the signaling molecules. the signaling molecules density reaches equilibrium around day 60.

Fig3 M.avi: the myofibroblasts. Around 120 days, the myofibroblast distribution reaches equilibrium.

Fig3 N.avi: the fibroblasts. The fibroblast distribution grows as follows. After a few days, when the displacement velocity density reaches equilibrium, the fibroblast distribution above the equilibrium decreases, and the fibroblast distribution below the equilibrium increases, except for the fibroblast distribution around the right boundary of the domain of computation, representing the center of the portion of skin that we model. The number of fibroblasts around this right boundary decreases until about 23 days, after which it increases towards equilibrium.

Fig3 rho.avi: collagen. The collagen density changes calmly: the density above the equilibrium moves downward to the equilibrium, and the density below the equilibrium moves upward to the equilibrium.

Fig3 v.avi: the displacement velocity. Initially, the displacement velocity density oscillates around zero, moving the mesh to the left and right. Shortly after the start of the simulation, the wave in the displacement velocity density fades out. Further, within approximately 15 minutes, the amplitude increases by a factor 10 above the equilibrium value, and by a factor 25 below the equilibrium value. Shortly after that, around approximately 1.5 hours, the amplitude of the displacement velocity density has increased by a factor 45, after which the amplitude decreases until zero.

Fig3 e.avi: the effective strain. The effective strain density oscillates around the (new) equilibrium.

All the constituents reach equilibria within 600 days, after which the distributions and densities do not change anymore. Both the displacement velocity density and effective strain density reach the equilibria within a few days, the displacement velocity density reaching the equilibrium  $v = 0$  first. Note that these results both confirm the non-monotonic convergence from the variations around  $\varepsilon$  (see Theorem 1 and Theorem 2). We see the mesh also moving in the plots of the constituents. While the displacement velocity density oscillates, the distributions and densities of the constituents move from the right to the left and back, until the distributions and densities move gradually towards the equilibria.

**Files corresponding to Figure 5.** The videos show the evolution of the constituents and the mechanics after perturbations in case the stability constraints are not met, but the the model still converges. Parameter values are  $\delta_c = 3 \cdot 10^{-4}$  and  $\mu = 100$ . We used a larger number of waves with  $k = 10$ .

Fig6 c.avi: the signaling molecules. Initially, the signaling molecule density decreases, but on approximately day 9 the upper bound of the density surpasses the initial upper bound (see the right plot in Figure 4). The signaling molecule density keeps increasing until day 215, affecting the (myo)fibroblast distributions and the collagen density, shown in Figure 5 (the videos).

Fig5 N.avi: the fibroblasts and Fig5 M.avi: the myofibroblasts. The initial perturbed waves in the (myo)fibroblast distribution fade out within 4.5 days. Both distributions move toward the corresponding equilibria  $10^4$  cells/cm<sup>3</sup> and approximately 0.16 cells/cm<sup>3</sup> (hence no cells), respectively. However, on days 63.5 and 65, for the fibroblasts and myofibroblasts respectively, the distributions move away from the equilibria. After the signaling molecule density decreases from day 215 on, the myofibroblast distribution, the collagen density and the

fibroblast distribution keep moving away from their equilibria until days 230, 250 and 260, respectively.

Fig6 rho.avi: collagen. The collagen density is not affected by this setup until day 120, after which this density increases. Collagen takes more time to fade out the initial perturbed waves.

From the moments where maxima and minima are reached, the distributions and densities of the constituents oscillate around a new equilibrium. At the end of the simulation of 1000 days the new equilibria in the center of the modeled skin are  $4.245 \times 10^{-11}$ , 9723, 76, and 0.1348 for the signaling molecules, the fibroblasts, the myofibroblasts, and collagen, respectively.

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

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