Tutorial 2: Elasticity – stretching, bending and amplification

Physics of Complex Systems M2 – Biophysics

1 Linear response

Here we investigate the relationship between macroscopic linear elasticity theory and two possible underlying microscopic models.

1.1 Affine deformation of a hyperstatic network

Consider a triangular lattice of harmonic springs with stiffness α in two dimensions. It behaves as an isotropic elastic solid on large length scales, and both its bulk and shear moduli can be expressed as functions of the stiffness α. Here we establish this relationship for the bulk modulus K.

1.1.1 Consider a deformation whereby the network is homogeneously and isotropically stretched, which results in a change of its volume V by δV. What is the form of the strain tensor γ associated with this deformation? Relate its components to δV.

1.1.2 Denoting by $r_i^{(a)}$ the initial position vector associated with vertex a, what is the displacement vector $u_i^{(a)}$ associated with the vertex following the deformation? Why is this type of deformation known as an "affine" deformation?

1.1.3 Write the work performed over the course of the deformation as a function of K and δV.

1.1.4 Write the microscopic expression for that same work, in terms of the spring stiffness α.

1.1.5 Deduce from this the relationship between K and α.

1.1.6 Generalizing this scaling to a hyperstatic 3D actin network of unspecified structure with bond length $s \simeq 400\,\text{nm}$, what typical value do you expect for its bulk modulus?

1.2 Bending-dominated elasticity of a hypostatic network

We now introduce another lattice, illustrated in Fig. 1. Note that despite its rather large unit cell, all its vertices are equivalent.

1.2.1 Will stretching interactions be enough to stabilize this network? Why? Drawing inspiration from the middle and right-hand-side of Fig. 1, illustrate its floppy mode of isotropic deformation.

1.2.2 Consider an isotropic overall deformation of the network as in Sec. 1.1. One possible response of the network to this kind of deformation is an affine deformation. How does the cost of such a deformation scale with the bond stiffness α?

1.2.3 Let θ be the angle between two consecutive bonds in a deformed network, as indicated in the right-hand-side of Fig. 1. We consider a mode of overall isotropic deformation whereby all hinges bend by the same angle θ. What is the new area of the unit cell?

1.2.4 We now introduce a bending interaction by associating an energy $e = k_B T \ell_p \theta^2/2s$ with each hinge between two consecutive bonds of length s misaligned by θ. How does the energy per unit cell depend on θ?

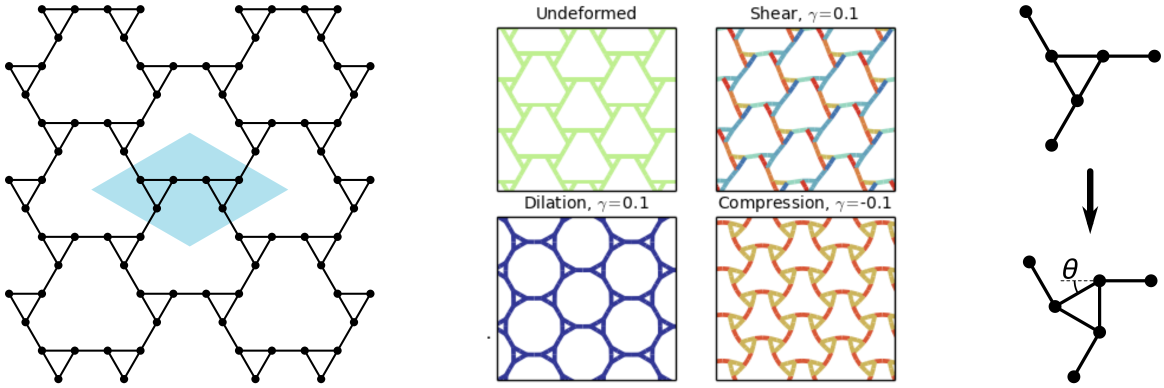

Figure 1: Simple bending-dominated lattice. *Left*: Ground-state structure with unit cell outlined in blue and vertices indicated by black circles. *Middle:* Numerically simulated deformations of a similar lattice. *Right:* Basic mode of network deformation considered here.

1.2.5 Assuming that the filaments constituting the network are much easier to bend than to stretch, how will the network deform in response to a uniform compression? Why is this known as a nonaffine deformation?

1.2.6 What is the bulk modulus of this network? Generalizing this scaling, what order of magnitude do you expect for the elastic moduli of an actin network?

1.3 Discussion

After examining these two examples, some conclusions are in order:

- Hypostatic networks avoid costly affine deformation by deforming along nonaffine soft modes.
- In the presence of a weak stabilizing interaction (*e.g.*, bending or prestress), these soft mode acquire a modulus without changing their spatial structure.
- The elasticity of hypostatic networks is nonaffine, yet linear at small deformations. In the case studied here it is bending-dominated (with a modulus $\propto k_BT\ell_p$ instead of $k_BT\ell_p^2$).

Although we computed bulk moduli because of the simplicity of the calculation involved, experimentalists virtually always report the shear moduli of their actin networks. Why?

2 Nonlinear response

2.1 Nonlinear bulk moduli

Beyond linear elasticity as explored in the main lectures, here we explore two possible microscopic causes for a nonlinear response at the material level.

2.1.1 *Constitutive nonlinearities.* Tutorial 1 showed us that a single bond in a network can respond nonlinearly to an applied force, for instance by buckling under compression and stiffening under extension. Using the force-extension relationship derived there, plot the pressure-volume relationship for the affinely deforming network of Sec. 1.1.

2.1.2 *Geometrical nonlinearities.* Plot the nonlinear pressure-volume relationship for the nonaffinely deforming network of Sec. 1.2. Discuss whether a nonlinear material response necessarily requires a material with an anharmonic Hamiltonian. If not, where does the nonlinearity come from?

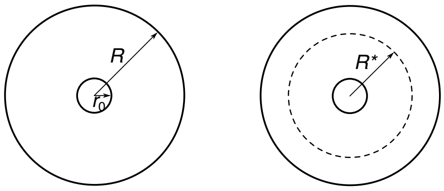

Figure 2: Geometry of our force transmission problem. *Left:* non-buckled case. *Right:* buckled case.

2.2 Active stress amplification by nonlinear materials

Contractile biological materials are typically composed of a fiber network with many actively contracting units embedded in it, be it molecular motors in the cytoskeleton or cells in an extracellular matrix. The contraction of the individual units induces a stress at the scale of the whole network, which is harnessed by the corresponding organism to perform a variety of functions. Here we assess how the elastic properties of the network affect this process of force generation in a very simplified spherical geometry. The force balance equation in this geometry reads

$$\frac{1}{r}\frac{\mathrm{d}}{\mathrm{d}r}\left(r\sigma_{rr}\right) - \frac{\sigma_{\theta\theta}}{r} = 0 \quad \text{in } d=2 \text{ dimensions} \tag{1a}$$

$$\frac{1}{r^2}\frac{\mathrm{d}}{\mathrm{d}r}\left(r^2\sigma_{rr}\right) - \frac{\sigma_{\theta\theta}+\sigma_{\phi\phi}}{r} = 0 \quad \text{in } d=3 \text{ dimensions} \tag{1b}$$

In addition, for a radial deformation $\mathbf{u}(r) = u(r)\hat{\mathbf{r}}$, the strain tensor in spherical coordinates is given by

$$\gamma = \begin{pmatrix} \partial_r u & 0 & 0 \\ 0 & u/r & 0 \\ 0 & 0 & u/r \end{pmatrix} \tag{2}$$

2.2.1 Rationalize the form of Eq. (1) by considering the balance of forces on a small $(\mathrm{d}r, \mathrm{d}\theta)$ element in two dimensions.

2.2.2 Solve the force balance equation for a linear elastic material and give the general form of the radial displacement $u(r)$ in a spherical geometry in arbitrary dimension d.

2.2.3 We consider a localized active unit exerting a radial stress discontinuity in $r = r_0$:

$$\lim_{\epsilon\to 0}\left[\sigma_{rr}(r_0+\epsilon) - \sigma_{rr}(r_0-\epsilon)\right] = \sigma_0 \tag{3}$$

with $\sigma_0 > 0$. Denoting by $\mathbf{f}$ the body force density exerted by the active unit on the network, what is the force dipole $D = \int(\mathbf{r}\cdot\mathbf{f})\,\mathrm{d}V$ exerted by this active unit on the network?

2.2.4 Now working in $d = 2$ for simplicity, we assume a fixed boundary $u(R) = 0$ at a radius $r = R$ as illustrated in Fig. 2. Give the form of the stress tensor everywhere in the medium, distinguishing the $r < r_0$ region from the $r_0 < r < R$ region.

2.2.5 How is the stress dipole exerted by the medium on the fixed boundary related to the active force dipole? This is actually just a specific instance of a much more general relation [1].

2.2.6 We now consider a nonlinear material that can only sustain compressive stresses up to a certain threshold σ_b, then buckles. In the buckled state, the compressive stresses saturate to $-\sigma_b$, while the extensile stresses can be arbitrary large. Assuming the material buckles in a region $r < R^*$ and then behaves linearly for $r > R^*$, give the stress tensor everywhere in the system in the limit $\sigma_0 \gg \sigma_b \Rightarrow R^* \gg r_0$.

2.2.7 Determine R^*. In this new nonlinear case, how is the boundary stress related to the active force dipole? Explain this stress amplification qualitatively.

References

[1] Pierre Ronceray and Martin Lenz. Connecting local active forces to macroscopic stress in elastic media. *Soft Matter*, 11(8):1597–1605, January 2015.