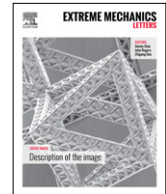




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Wrinkling, creasing, and folding in fiber-reinforced soft tissues

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ABSTRACT

Many biological tissues develop elaborate folds during growth and development. The onset of this folding is often understood in relation to the creasing and wrinkling of a thin elastic layer that grows whilst attached to a large elastic foundation. In reality, many biological tissues are reinforced by fibers and so are intrinsically anisotropic. However, the correlation between the fiber directions and the pattern formed during growth is not well understood. Here, we consider the stability of a two-layer tissue composed of a thin hyperelastic strip adhered to an elastic half-space in which are embedded elastic fibers. The combined object is subject to a uniform compression and, at a critical value of this compression, buckles out of the plane – it wrinkles. We characterize the wrinkle wavelength at onset as a function of the fiber orientation both computationally and analytically and show that the onset of surface instability can be either promoted or inhibited as the fiber stiffness increases, depending on the fiber angle. However, we find that the structure of the resulting folds is approximately independent of the fiber orientation. We also explore numerically the formation of large creases in fiber-reinforced tissue in the post-buckling regime.

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1. Introduction

In recent years there has been considerable interest in the pattern created by the growth of a thin elastic layer attached to an elastic foundation [1]. The generic behavior of such systems is to respond to a mismatch in stresses between the two layers by wrinkling (so that stress can be relaxed in the stiffer layer). However, the development of this wrinkling pattern beyond the onset of instability is surprisingly intricate: for large ratios of layer μ_l to foundation μ_s stiffness, $\mu_l/\mu_s \gtrsim 10$, a period-doubling instability occurs [2–4] due to nonlinearities in the substrate

response. For small ratios of layer to foundation stiffness, $\mu_l/\mu_s \lesssim 10$, the system instead localizes the deformation and a fold or crease develops. For many biological systems, it is the latter scenario that is of most interest; for example, the deep folding patterns that are formed during the growth of brains are believed to be partially caused by this instability [5–7].

While the elastic instability of a growing multilayer material gives rise to wrinkling and folding patterns that appear similar to those observed *in vivo*, the material that makes up the white matter of the brain is known to be highly anisotropic, consisting of pre-stretched, axonal fiber bundles [8,9]. It is not clear whether and how this anisotropy might impact the relatively simple elastic behavior discussed above. Studies in developing chick embryos [10] indicate that the fibrous structure is relatively

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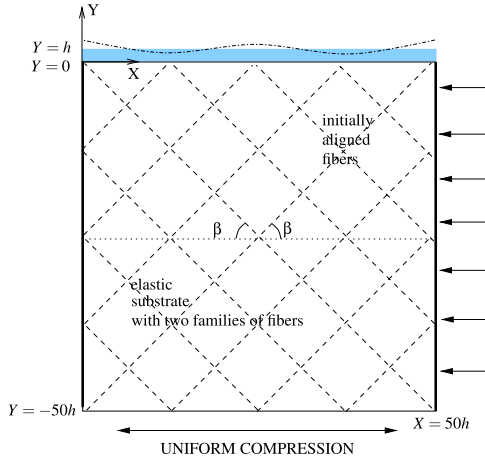


Fig. 1. Setup of the mathematical model.

passive and, further, that the fiber orientation is a consequence of the folding pattern, rather than its cause. Similarly, many other biological tissues are reinforced with collagen fibers, such as tendons and ligaments [11], annulus fibrosus in the spinal cord [12] and arterial walls [13–15].

In this paper we consider a model system that is motivated by the various tissues in the brain during development: a slab of homogeneous elastic tissue (representing the white matter) is connected to a thin layer of a stiffer tissue (representing the cerebral cortex). The setup is shown schematically in Fig. 1 and is similar in spirit to other models of cerebral cortex folding [16,17]. In each region, we assume that the material is elastic with reinforced fibers and a fiber pre-stretch. We study the influence of the fiber orientation and of the pre-stretch by direct computational simulations. We then compare these results with those of a linear stability analysis.

2. The model

In the reference configuration, the two-layer material is described by the coordinates $\mathbf{X} = (X, Y, Z)$, shown in Fig. 1, with the interface between the two layers is denoted $Y = 0$. Deformation of the tissue to a new configuration parametrized by current coordinates $\mathbf{x} = (x, y, z)$, is described by the mapping $\mathbf{x} = \chi(\mathbf{X}, t)$, where t is time, with corresponding deformation gradient tensor $\mathbf{F}(\mathbf{X}, t)$. Both layers of tissue are assumed hyperelastic, and can be described by strain–energy functions $\mathcal{W}_l(\mathbf{F})$ and $\mathcal{W}_s(\mathbf{F})$, for the upper layer and the substrate, respectively. We assume that the deformation occurs sufficiently slowly that inertial effects can be neglected and that the strain lies in the (X, Y) -plane.

The upper layer is modeled as a neo-Hookean material of uniform thickness H with shear modulus μ_l and bulk modulus K_l , i.e.

$$\mathcal{W}_l = \mu_l(\bar{\lambda}_1^2 + \bar{\lambda}_2^2 + \bar{\lambda}_3^2 - 3) + \frac{1}{2}K_l \log J, \tag{1}$$

where $J = \det(\mathbf{F})$ and $\bar{\lambda}_j$ are the principal values of the left Cauchy–Green tensor $\bar{\mathbf{F}}\bar{\mathbf{F}}$, where $\bar{\mathbf{F}} = J^{-1/3}\mathbf{F}$. The material is modeled as a standard fiber-reinforced material.

Table 1

Benchmark parameters used in the computational simulations. All moduli are dimensionless.

Variable	Symbol	Benchmark value
Fiber volume fraction	φ_f	0.1
Matrix volume fraction	φ_m	0.9
Upper layer bulk modulus	K_l	$3\mu_l = 30$
Substrate bulk modulus	K_s	3
Upper layer shear modulus	μ_l	10
Fiber-stiffness	μ_f	10
Fiber pre-stretch	λ_f	1.0

That is, the substrate is modeled as a neo-Hookean elastic half-space, in the region $Y < 0$. This half space contains two families of fibers which are assumed to contribute to the strain–energy density at the lowest possible positive powers in the fiber strain. For simplicity, we assume that the matrix and fibers experience the same deformation gradient, so the energy of the substrate may thus be written using an additive decomposition in the form $\mathcal{W}_s = \varphi_m \mathcal{W}_m + \varphi_f \mathcal{W}_f$ where

$$\mathcal{W}_m = \mu_s(\bar{\lambda}_1^2 + \bar{\lambda}_2^2 + \bar{\lambda}_3^2 - 3) + \frac{1}{2}K_s \log J, \tag{2}$$

$$\mathcal{W}_f = \sum_{i=1}^2 f(I_4^{(i)}), \quad f(\eta) = \frac{\mu_f}{4}(\eta - \lambda_f^2)^2. \tag{3}$$

Here φ_f and φ_m represent the volume fractions of all fibers and matrix, respectively, while

$$I_4^{(i)} = (\mathbf{N}^{(i)})^T \mathbf{F}^T \mathbf{F} \mathbf{N}^{(i)}, \quad (i = 1, 2),$$

where the unit vector $\mathbf{N}^{(i)}$ ($i = 1, 2$) is the direction in which fiber family (i) is aligned in the reference configuration. We have therefore assumed that the fibers are equal and opposite (with angle $\pm\beta$ with the X -direction). Finally, λ_f is the fiber-prestress, which is assumed constant. For $\lambda_f < 1$, the fibers are under tension in the reference configuration. It should be noted that this constitutive model allows the fibers to bear compressive loading [18], which is usually neglected when modeling tissues reinforced by collagen fibers such as arteries [14].

We non-dimensionalize all lengths by the thickness of the upper layer, H , and all moduli by μ_s , i.e. we take $\mu_s = 1$ without loss of generality. Using a Poisson ratio for both layers of approximately 0.35 [19], we take the bulk moduli $K_s \approx 3, K_l \approx 3\mu_l$. The values of other fixed parameters are based on those relevant for brain tissues and are given in Table 1.

Growth of the material is mimicked by uniform compression of the two-layer material parallel to the X direction. The key parameter controlling this compression is the end-shortening $d = \Delta L/L$.

2.1. Methods

The model is solved implicitly using an ABAQUS UMAT, which for a given deformation gradient tensor \mathbf{F} requires both the corresponding Cauchy stress tensor $\boldsymbol{\sigma}$ and a stiffness tensor \mathbf{C} . The first Piola stress tensor for the neo-Hookean component of each layer is computed using a predictor–corrector method used previously by [20], based

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