Surface and interfacial creases induced by biological growth are common types of instability in soft biological tissues. This study focuses on the criteria for the onset of surface and interfacial creases as well as their morphological evolution in a growing bilayer soft tube within a confined environment. Critical growth ratios for triggering surface and interfacial creases are investigated both analytically and numerically. Analytical interpretations provide preliminary insights into critical stretches and growth ratios for the onset of instability and formation of both surface and interfacial creases. However, the analytical approach cannot predict the evolution pattern of the model after instability; therefore nonlinear finite element simulations are carried out to replicate the poststability morphological patterns of the structure. Analytical and computational simulation results demonstrate that the initial geometry, growth ratio, and shear modulus ratio of the layers are the most influential factors to control surface and interfacial crease formation in this soft tubular bilayer. The competition between the stretch ratios in the free and interfacial surfaces is one of the key driving factors to determine the location of the first crease initiation. These findings may provide some fundamental understanding in the growth modeling of tubular biological tissues such as esophagi and airways as well as offering useful clues into normal and pathological functions of these tissues.

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I. INTRODUCTION

Growth and remodeling of a developing soft biological tissue are two highly complex processes which have crucial effects in the normal development and pathological status of a biological tissue [1]. It has been shown that mechanical factors have considerable influence on the growth and remodeling of growing structures beside the biological and genetic factors [1–4]. Generally, biological tissues are composed of multiple layers of different thicknesses, material properties, and growth rates, e.g., the skin [5], brain [6], artery [7], gut [8], and esophagus [9]. Nonuniform growth results in the appearance of strain mismatch among the layers and leads to the advent of residual stresses [10]. This stress is created due to the integrity of the biological structures preventing self-overlap or overlap between tissues [11–14]. Residual stress is also believed to play a crucial role in morphogenesis and regulation of the material properties of biological systems [12,15–17]. It has been proven that once the compressive residual stress exceeds a critical value, in order to release its potential energy the tissue buckles into a new configuration [12]. Beyond the critical condition, three common types of morphological instability can be observed: wrinkling, folding, and creasing [1]. In contrast to wrinkling and folding phenomena, creasing in soft biological tissues (especially the interfacial creasing) has not been well studied and addressed appropriately. Therefore, it is necessary to find a systematic approach to analytically and numerically quantify crease formation in the soft biological tissues.

Creases with sharp edges usually form in soft materials without a hard skin when beyond a certain critical value of compression, depending on material properties [18–21]. One of the main characteristics of creases is the development of the self-contact phenomenon after instability. Creases bifurcate locally in space and are large in amplitude, in contrast to wrinkles which bifurcate nonlocally in space and are comparatively minuscule in amplitude. Studies show that a flat and soft slab, after having a critical compressive strain applied, starts to develop creases, and the anisotropy of applied strains controls the pattern of creases [22]. Critical strain for the onset of surface creases in a single layer is lower than that for the onset of surface wrinkles [23]. However, it should be mentioned that for a bilayer structure with comparable elastic moduli for both layers, the critical strain required for the onset of creases can be either smaller or larger than that for the onset of wrinkles. It depends on ratios of the moduli and thicknesses of both layers [24]. It has been shown that during growth, multilayer hyperelastic soft tissues with considerable differences in the shear moduli of each layer tend to develop wrinkles [1,9], while the same structures using layers with material properties more similar to each other prefer to develop creases [25–27]. Beyond the simple sinusoidal wrinkling, new complex morphologies emerge in a multilayer structure being compressed or grown, e.g., period doubling and period quadrupling [9]. A recent study showed that this kind of instability can also be observed in a low stiffness regime and that pattern formation is highly sensitive to small imperfections [28]. It is worthwhile mentioning that classical linear perturbation analysis is able to predict the critical condition for the onset of wrinkles, whereas it fails to determine the critical condition for the onset of creases [29]. Generally, creases may be divided into two types: surface creases and interfacial creases. Although the formation mechanisms of both kinds of creases are almost the same, there are still a few differences between the critical strain and shape selection [30]. A surface with a neo-Hookean incompressible material under compression can generate creases on its free surface when the stretch ratio (normal to the tangential) reaches a critical amount; this critical value does not depend on the shear modulus of the material [29]. However, for the onset of an interfacial crease between two bonded neo-Hookean...
materials, the critical compressive strain depends on the shear modulus ratio of the two layers [30]. In a surface crease, the material develops a self-contact pattern, but in an interfacial crease the presence of two materials prevents the interface from self-contact, so this kind of crease is V shaped [30]. A recent experimental study has shown that interfacial creases always form at a lower compression than interfacial wrinkles do [30]. Figure 1 shows mucosal wrinkling of a bovine esophagus. Since the mucosa is much stiffer than the submucosa, the mucosa forms a wrinkled pattern on the submucosa. Interfacial creases also can be detected in the interface between the submucosa and muscle which has not been addressed sufficiently. Indeed, it is worth exploring the mechanism and shape selection of interfacial creases in soft tissues.

Few types of studies have been done related to surface creases, especially in the case of interfacial creases in the biological tissues, whereas wrinkling and folding phenomena in growing multilayer biological tissues have been studied widely [31–35]. The main goal of this study is to develop a computational mechanical model to seek answers to what factors contribute to the formation of surface and interfacial creases in a developing bilayer biological tissue. In what follows, we also strive to answer these two intriguing questions: How do contributing factors control the priority of the structure morphology to produce surface or interfacial creases and why are surface creases more common than interfacial creases in biological tissues? In order to answer these, we will construct plane-strain tubular bilayer structures with varying thicknesses, growth ratios, and material properties. Both theoretical and computational approaches will be utilized to determine deformation and stress fields of the growing structure and present the surface morphology after instability. Findings from this study are applicable to interpret the interfacial creases in the tubular multilayer organs like esophagi or invagination of a soft tissue in an elastic environment and the creation of villi in the gut of various species [8].

FIG. 1. Wrinkling of mucosa and interfacial creases in the esophagi of the bovine. Arrows show the sites of interfacial creases. Figure reprinted from Ref. [9] with permission.

FIG. 2. Initial and current states of a growing structure. The deformation gradient (F) is decomposed into a growth tensor (G) and an elastic tensor (A).
Many biological soft tissues can be modeled by a hyperelastic material with a strain energy function \( W(A) \). The Cauchy stress \( \sigma \) is related to the strain energy function by [12]

\[
\sigma = \lambda A \frac{\partial W}{\partial A} - p I,
\]

where \( p \) is the Lagrangian multiplier to ensure the incompressibility condition and \( I \) is a second-order unit vector. In the absence of any body force, mechanical equilibrium imposes

\[
\text{div} \ \sigma = 0,
\]

where “div” stands for the divergence operator in the current configuration. There are several proposed material behaviors for hyperelastic materials [36]; here a simple and common model, isotropic nonlinear neo-Hookean, is implemented.

\[
W = \frac{\mu}{2} (\lambda_r^2 + \lambda_\theta^2 + \lambda_z^2 - 3),
\]

where \( \mu \) is the shear modulus and \( \lambda_r, \lambda_\theta, \lambda_z \) are the radial, circumferential, and axial principal stretches, respectively.

Consider a tubular soft bilayer with layers of different material properties growing within a rigid confinement. The outer layer of the tissue is considered to be fixed as shown in Fig. 2. This ideal assumption is considered to mimic the confining effect of a surrounding environment. The inside and outside radii of the inner layer are \( A \) and \( B \), and the inside and outside radii of the outer layer are \( B \) and \( C \). The initial and undeformed configuration for the tube is defined by

\[
X = (R, \Theta, Z)
\]

\[
A \leq R \leq C, \quad 0 \leq \Theta \leq 2\pi, \quad 0 \leq Z \leq L,
\]

where \( R, \Theta, \) and \( Z \) are cylindrical coordinates in the reference state. \( L \) is the longitudinal length of the tube. \( B \) is the interface radius between the two bonded layers. Due to the biological growth, the tube deforms axisymmetrically before the occurrence of instability. Growth may occur in the inner layer, outer layer, or both of them. The new and current configuration after growth is defined by \( x = (r, \theta, l) \),

\[
a \leq r \leq c, \quad 0 \leq \theta \leq 2\pi, \quad 0 \leq z \leq l,
\]

where \( r, \theta, \) and \( z \) are cylindrical coordinates in the deformed state and \( l \) is the deformed axial length of the tube. \( b \) is the interface radius after the growth and deformation. In the case of axisymmetric and plane-strain deformation, the deformation field after growth is just a function of the radius, \( r = r(R) \), so, the circumferential and longitudinal coordinates in both undeformed and deformed states stay the same.

For isotropic growth, the growth tensor can be characterized by \( G = g I \), where \( g > 1 \) is for growth, \( 0 < g < 1 \) represents atrophy, and \( I \) is the unit tensor. But by the assumption of plane-strain conditions without deformation or growth in the axial direction, \( g_z \) is considered to be unit.

### B. Numerical method

A layered computational model based on the nonlinear finite element (FE) method with isotropic growth is implemented to capture realistic morphologies of a biological structure after the onset of instability. The model of both the inner and outer layers is considered to be a neo-Hookean hyperelastic material, and growth is simulated via thermal expansion [18,37]. The free surface of the inner layer is allowed to self-contact. The fixed boundary condition is applied in the outer radius of the outer layer. DYNAMIC-EXPLICIT solver in the commercial software ABAQUS (version 6.13), which is suitable for large deformation, nonlinear, and quasistatic problems, is implemented to depict pattern changes in the model. Both layers mesh with the plane-strain elements where different mesh sizes are used to ensure the robustness of the simulation results and mesh independence. Following the incompressibility we used in this biological structure, the growth ratio of the layer \( (g_i) \) can be defined as the square root of the ratio of the deformed surface area \( S_i \) to the initial surface area \( S_{0i} \), i.e., \( g_i^2 = S_i / S_{0i} \), where \( i = 1 \) represents the inner layer and \( i = 2 \) the outer layer.

### III. RESULTS AND DISCUSSIONS

#### A. Deformation field and residual stress

By applying the deformation gradient in a cylindrical coordinate for the presented model in Fig. 2 and from Eq. (1), the elastic deformation tensor can be extracted,

\[
A = \begin{pmatrix} \lambda_r & 0 & 0 \\ \lambda_\theta & 1 & 0 \\ \lambda_z & 0 & 1 \end{pmatrix}, \quad A_2 = \begin{pmatrix} g^{-1} \lambda r & 0 & 0 \\ 0 & 1 & 0 \\ 0 & 0 & 1 \end{pmatrix}
\]

In which \( \lambda_i \) is the principal stretch. With the incompressibility constraint \( \det A = 1 \), we have

\[
\frac{r}{R} \frac{\partial r}{\partial R} = g^2.
\]

Integration of Eq. (8) on the boundary of two layers with imposed boundary conditions gives the deformation field of the structure

\[
r^2 - a^2 = g_1^2 (R^2 - A^2) \quad \text{for} \quad A \leq R \leq B,
\]

\[
r^2 - C^2 = g_2^2 (R^2 - C^2) \quad \text{for} \quad B \leq R \leq C,
\]

In which \( g_1 \) and \( g_2 \) are isotropic growth ratios for the inner layer and outer layer, respectively. Boundary conditions are fixed boundary in \( C \) and continuity in the interfacial radius between two layers \( (R = B) \). By continuation in the interface, the deformed inner radius \( a \) of the structure is

\[
a = \left[ C^2 - g_2^2 (C^2 - B^2) - g_1^2 (B^2 - A^2) \right]^{1/2},
\]

and the deformed interface radius \( b \) is

\[
b = \left[ C^2 - g_2^2 (C^2 - B^2) + g_1^2 (B^2 - A^2) \right]^{1/2}.
\]

Equations (9)–(12) describe the deformation of the tubular structure induced by growth. For preventing self-contact at the inner radius of the structure after growth, the isotropic growth ratios should satisfy \( a > 0 \).

Stretch ratios have been demonstrated as a determining factor to trigger instability in soft materials [18,29]. For the sake of further implementation, it is necessary here to detail their derivations. Let \( \lambda = \lambda_\theta \); the assumption \( \det A = 1 \) leads...
where the subscript 1 is for the inner layer and 2 is for the outer layer. $\lambda_2$ is just a function of the growth ratio in the outer layer, whereas $\lambda_1$ is a function of both growth ratios in the inner and outer layers. Based on Eqs. (2) and (4) the Cauchy stress components are derived as

$$\sigma_{rr} = \mu \lambda_1^{-2} - p, \quad \sigma_{\theta \theta} = \mu \lambda_2^2 - p.$$  

The equilibrium equation, Eq. (3), is derived as

$$\frac{\partial \sigma_{rr}}{\partial r} + \frac{\sigma_{rr} - \sigma_{\theta \theta}}{r} = 0.$$  

After derivations, with Eqs. (13)–(16) the stress distribution can be expressed for the inner layer as

$$\sigma_{rr1} = \frac{\mu_1}{2} \left[ \lambda_1^{-2} - \lambda_{a1}^{-2} + \ln \left( \frac{\lambda_{a1}^2}{\lambda_1^2} \right) \right] A \leq R \leq B,$$

$$\sigma_{\theta \theta1} = \sigma_{rr1} + \mu_1 (\lambda_1^2 - \lambda_{a1}^{-2}) A \leq R \leq B,$$

where $\mu_1$ is the shear modulus of the inner layer and $\lambda_{a1} = a/g_1 A$. Using the same approach for the outer layer gives

$$\sigma_{rr2} = \sigma_{rr1} \left. \right|_{\lambda_{a1} = \lambda_{b1}} + \frac{\mu_2}{2} \left[ \lambda_2^{-2} - \lambda_{b2}^{-2} + \ln \left( \frac{\lambda_{b2}^2}{\lambda_2^2} \right) \right]$$

for $B \leq R \leq C,$

$$\sigma_{\theta \theta2} = \sigma_{rr2} + \mu_2 (\lambda_2^2 - \lambda_{b2}^{-2})$$

for $B \leq R \leq C,$

where $\mu_2$ is the shear modulus of the outer layer, $\lambda_{b1} = b/g_1 B$, and $\lambda_{b2} = b/g_2 B$.

The thickness of layers is variable so as to be able to parametrically capture thickness effects on the growth, instability, and remodeling of the tubular bilayer. For a special case, $A = 1$, $B = 1.2$, and $C = 2$; Fig. 3 shows the deformation field and stretches ($\lambda$) for a growing bilayer structure with two different growth cases.

For the sake of simplicity, results are mapped to the initial configuration of the structure. As can be noticed from Fig. 3, the deformation field between the inner and outer layers is continuous as expected, but stretches at the interface are not continuous and have jumps. The higher growth ratio in the outer layer leads to more deformation and higher stretches in both layers. It is also noteworthy to mention that stretch is not a function of the material properties of the layers but rather a function of their geometry and growth.

Figure 4 illustrates that in the absence of external loads, growth induces residual stresses in both layers. Representative parameters are $\mu_2/\mu_1 = 10$, $A = 1$, $B = 1.2$, $C = 2$, $g_1 = 1$, and $g_2 = 1.05$. Cauchy radial and circumferential stresses are normalized by the modulus ratio of the inner layer; $\bar{\sigma}_{rr} = \sigma_{rr}/\mu_1$ and $\bar{\sigma}_{\theta \theta} = \sigma_{\theta \theta}/\mu_1$.

Due to the biological growth, the maximum circumferential stress occurs on the free surface of the inner layer [19]. Compressive circumferential stress is observed to be discontinuous at the interface with a lower magnitude in contrast to the stress at the free surface. Several previous studies have revealed that beyond a critical point, compressive stresses in the free surface or interface of soft materials may lead to the formation of creases [18,25,26,29,30,38]. These compressive stresses may play an important role in the instability and shape evolution of the model.

**B. Stretch ratio and instability**

In order to find the critical strain for the onset of surface or interfacial creases in an incompressible neo-Hookean material, creasing instability has been analyzed by comparing the elastic energy in a creased elastomer and that in a smooth elastomer [29,30]. Results of these studies show that the critical strain for onset of creases on the free surface is $\varepsilon_c \approx 0.35$ and for the interfacial creases it is a function of the modulus ratio of the layers. In both surface and interfacial creases, critical strains are independent of any length scale. In fact, the surface crease is a special case of interfacial crease in which the modulus ratio of the stiff layer to the soft

![FIG. 3. Deformation field and stretches. $A = 1$, $C = 2$, and the interface is at $B = 1.2$.](image-url)

![FIG. 4. Normalized radial and circumferential stresses in the growing structure. $A = 1$, $C = 2$, and the interface layer is at $B = 1.2$. Growth only takes place in the outer layer, $g_2 = 1.05$.](image-url)
layer is infinite. The critical strain for the onset of interfacial creasing decreases as the modulus ratio of the stiff layer to the soft layer increases [30]. Calculation has shown that when an incompressible neo-Hookean material in the plane-strain condition is compressed to a critical point, the normal to tangential stretch ratio is close to 2.4 [29], equivalent to the critical strain \( \varepsilon_c \approx 0.35 \). This beneficial number has been used in several studies to predict critical growth ratios for the onset of surface creases in growing structures [18,19,39]. However, the mechanism and criteria for the onset of surface and interfacial creases in biological tissues have not been addressed very well. In what follows, we are going to unravel these issues.

Following the idea of the critical stretch ratio, in our model for the onset of surface creases in the inner layer, the radial to circumferential stretch ratio should satisfy \( \lambda_r/\lambda_\theta \geq 2.4 \). But for the onset of interfacial creases between the inner and outer layer this critical stretch ratio is assumed to be a function of their modulus ratio, i.e., \( \lambda_r/\lambda_\theta = f(\mu_2/\mu_1) \). Figure 5 shows the dependency of the critical stretch ratio \( \lambda_r/\lambda_\theta \) on the shear modulus ratio of the two bonded layers for the onset of interfacial creases which have been derived from Ref. [30]. Here we will focus on models with a higher shear modulus in the outer layer than in the inner layer. If the inner layer of a structure has a higher shear modulus ratio than the outer layer, it will typically generate wrinkles in a stiff layer on a soft substrate, a phenomenon which has been well reported in many studies [31,34,40,41].

Figure 5 shows that the critical stretch ratio for the onset of interfacial creases decreases as the modulus ratio of the bilayer increases. In the limiting case \( \mu_2/\mu_1 = \infty \), the critical stretch ratio approaches the critical one in surface creases, \( \lambda_r/\lambda_\theta \approx 2.4 \). When the shear modulus ratio is close to unit, two layers can be considered as a uniform hyperelastic material in which it is difficult to initiate the interfacial crease even in high compressive strains, \( \varepsilon_c \approx 1 \). The presence of the surface creases on the inner layer or interfacial creases depends on the competition of the critical stretch ratio \( \lambda_r/\lambda_\theta \) between the inner surface and the interface of the bilayer structure. With the definition \( \lambda = \lambda_\theta \) and \( \lambda = \lambda_\theta^{-1} \) the radial to circumferential stretch ratio is \( \lambda_r/\lambda_\theta = 1/\lambda^2 \) where \( \lambda \) for both inner and outer layers has been derived in Eqs. (13) and (14). Figure 6 depicts the radial and circumferential stretches and their ratio in a growing structure with \( A = 1, B = 1.2, C = 2 \), and \( g_2 = 1.05 \). The competition between the stretch ratios in the free and interfacial surfaces is one of the key driving factors to determine the location of the first crease initiation. The modulus ratio between two layers also plays an important role in the determination of the crease formation. By comparing the data from Fig. 6 with the critical values from Fig. 5, we can find the critical growth ratios for specific cases to start instability and crease formation as well as the location of crease formation.

Equations (13) and (14) imply that the initial interfacial geometry in each layer can affect the stretch ratio during the deformation. Figure 7 shows the dependency of the stretch ratio for each layer on its geometry with the structural parameters \( A = 1 \) and \( C = 2 \) as well as the growth ratios \( g_1 = 1 \) and \( g_2 = 1.1 \). There are two different cases: one with the interface at \( B = 1.5 \) and the other one at \( B = 1.8 \).

From Fig. 7, it can be noticed that as the position of the interface changes, the stretch ratio responds accordingly in both layers. A higher thickness of the outer layer leads to
Figure 9(a) clearly states that if growth just occurs in the inner layer of the structure, there are no stretches in the outer layer. This result shows that growth in the inner layer only has the potential to create surface creases but not interfacial creases. The higher the growth ratio of the inner layer, the higher the stretch ratio the free surface experiences. Figure 9(b) illustrates that if growth just takes place in the outer layer, a significant stretch ratio can be observed even on the free surface of the inner layer. Therefore, it is expected that by the growth of the outer layer, surface creases can be produced on the free surface. Moreover, the interface may also experience a considerable stretch ratio which can lead to the formation of interfacial creases. Figure 9(c) shows an example of the case in which growth takes place in both layers, wherein high stretch ratio can be observed in both free and interfacial surfaces. The dotted line in Fig. 9(c) indicates that both the inner and outer layers have the same growth ratio which makes the stretch ratio continuous across the interface. The behaviors of the third case in Fig. 9(c) can roughly be mimicked by the second case through considering more growth in the outer layer. Also, the second case can cover both surface and interfacial creases which just depend on a single variable, the growth ratio in the outer layer, \( g_2 \). Therefore, for simplicity we would like to focus on the model based on the second case from Fig. 9(b) to investigate the critical growth ratio and the morphological evolution after instability in the following sections.

C. Crease formation and critical growth ratio

It was found that the stretch ratio and the material properties of the layers are determining factors to initiate surface and interfacial creases in a soft tubular bilayer. The maximum value of the stretch ratio in the free or the interfacial surface is determined by the initial geometry and growth ratio of the bilayer structure. It is not easy to calculate the growth ratio of layers in vivo; therefore we dynamically increase the growth ratio from a unit to the value where the system initiates instability and creasing. Pathological disorders affect the growth ratio and overgrowth of layers may lead to obstruction in organs such as airways and esophagi \([9,32,33]\).

Here we want to show how surface and interfacial creases are developed step by step through a special case. Figure 10 shows the dependency of the stretch ratio \((\lambda_r/\lambda_θ)\) at the free surface and interface on the growth ratio of the outer layer. For this case the interface radius is \(B = 1.2\) and both free
surfaces on the growth ratio. Growth only takes place in the outer layer, four highlighted regions can be determined. After growth, the structure could be in a flat state or develop surface creases and then form interfacial creases, or develop interfacial creases and then surface creases.

It is evident from Fig. 11 that when the inner layer thickness is below \( \approx 1.9 \) creasing always occurs in the free surface first, accompanied by interfacial creasing later. Another intriguing observation is that an unusually high biological growth in a structure with a very thin outer layer is needed to trigger creases in the free surface while interfacial creases may happen earlier. It should be mentioned that all findings so far are related to the initiation of the creases based on the theoretical approach in which the growing bilayer structure is assumed to keep its axisymmetric pattern under any growth ratio. In the next section, nonlinear finite element analysis will be performed to show the postsecondary morphological evolution after the system reaches the critical growth ratio predicted from the theoretical method.

**D. Computational results**

Figure 12 shows the morphological evolution of a growing bilayer structure with \( B = 1.4 \). As discussed in Fig. 11, with the growth in the outer layer the stretch ratio in both layers grows and changes the pattern of the structure. Figure 12(a) shows the initial geometry of the model without growth. With growth in the outer layer, the structure starts to expand symmetrically [Fig. 12(b)] and after a certain value of growth ratio, the model loses its stability and develops surface creases first as shown in Fig. 12(c). With the continuation of growth, interfacial creases are observed at the interface of the two layers, as depicted in Fig. 12(d). Since the strain is identical at any place on the free surface under the axisymmetric deformation, any point on the free surface can attain the critical conditions necessary to start creasing. Therefore, the position and number of creases on the free surface cannot be perfectly determined by the critical condition for the initiation of creases, which is also valid for interfacial creases. However, pattern selection and the number of creases on the free surface or on the interfacial surface can be attributed to the process of minimizing the strain energy in the structure [18].
where $t$ is the thickness of the film on the substrate before compression and $\epsilon$ is the compressive strain beyond the critical value [27,42]. By replacing $\epsilon$ with $\lambda$, Eq. (24) is represented by

$$\omega_{\text{crease}}/t \approx 3.5(1 - \epsilon),$$

(24)

where $\omega_{\text{crease}}/t$ is the wavelength of creases on the free surface can be calculated approximately and compared with numerical results. Equation (24) is applicable when a uniform compressive strain is applied through the thickness of the film, whereas in the model of interest the strain and stretch through the thickness of inner layer is nonuniform as shown in Fig. 3. Compresive strain in the free surface of the inner layer is higher than the one at the interface.

Since strain at the free surface is a more effective factor in determining the formation of surface creases, it can be estimated from Eq. (24). From Eq. (13) and with $g_2 = 1.2$, the stretch ($\lambda$) in the free surface can be determined as 0.320 which implies the dimensionless wavelength is

$$\omega_{\text{crease}}/t \approx 1.12.$$  

From the computational model in Fig. 12(c), this ratio is calculated as $\omega_{\text{crease}}/t \approx 1.10$. In the computational model the wavelength of creases is considered as a distance between two walls of a crease and measured from their middle line. The theoretical estimated value and computational result are in good agreement with each other, but it should be mentioned that the stretch ($\lambda$) from Eq. (13) is valid until the initiation of instability and after that critical point or at higher growth ratios it is no longer applicable. When the growth ratio increases, compressive strain builds up consequently in the inner layer and the wavelength of creases decreases as depicted in Fig. 12(e). Equation (24) is just extracted for surface creases and cannot be used for the calculation of the interfacial crease wavelength. Depth of the creases is also a linear function of the applied compressive strain [27], a large strain corresponding to a deep crease as seen in Fig. 12(e). It is intriguing to see that the sharp crease in the interfacial creases is formed towards the outer layer where the material is stiffer compared with the inner layer [30].

Our model proves that surface creases develop a self-contact pattern, but the presence of two materials in an interfacial crease prevents the interface from self-contact; therefore it forms a V shape.

Critical growth ratios for the onset of both surface and interfacial creases extracted from FE analysis are compared with the theoretical findings in Fig. 11. Generally, there is good agreement between theoretical and finite element results. Since the outer layer of the structure is stiffer than the inner layer, before crease formation the structure remains symmetric in the FE analysis as the theoretical assumption states. This explains why good agreement is achieved between the theoretical and numerical results of the structure with a thin inner layer. For the structure with a thick inner layer, e.g., $B = 1.9$, interfacial creases initially developed, followed by the free surface creases as shown in the top row of Fig. 13.

At the beginning the number of interfacial creases is four, but with further growth more creases can be observed at the interface.

FIG. 12. (a–e) Step by step morphological evolution of a growing bilayer. Growth just takes place in the outer layer and $A = 1$, $B = 1.4$, $C = 2$, $\mu_2/\mu_1 = 10$. Fig. 12, the free surface develops eight creases and with the continuation of growth they become deeper and sharper. After a certain amount of growth, four interfacial creases develop on the interface and then evolve into eight. This biological structure can continue to grow until the inner layer completely fills the free space of the structure.

Previous studies on the compressed flat bilayer structure show that beyond the critical strain ($\epsilon_c = 0.35$) a flat surface develops creases with wavelength $\omega_{\text{crease}}$:

$$\omega_{\text{crease}}/t \approx 3.5(1 - \epsilon),$$

(24)
FIG. 13. Morphological evolution of a growing bilayer structure with different initial interfacial radii. Time steps from 1 to 4 show the evolution of morphology of the model (growth ratios are not the same in columns). Growth only takes place in the outer layer and $A = 1$, $C = 2$, $\mu_2/\mu_1 = 10$.

earlier than the ones with lower shear modulus ratio. When the shear moduli of two layers are close to each other it is difficult to observe interfacial creases but there is evidence of a wrinkled pattern as shown in the bottom row of Fig. 14. Since the thickness ratio of the inner layer to the outer layer is the same for all three cases, the number of surface creases in all cases also remains the same as evidenced in other papers [1,18].

But for interfacial creasing, the number of creases is also related to the shear modulus ratio. As expected, in Fig. 14 the number of interfacial creases is different for these three cases, and the model with a higher shear modulus ratio shows deeper and sharper creases than the model with a lower shear modulus does. For the ideal model with infinite shear modulus ratio, interfacial creases will be in a self-contact pattern as surface creases. Our results showed that when a soft layer grows on a stiffer layer, interfacial creases may be observed.

As a proof for the results, a recent study in a flat bilayer structure with close stiffness of layers showed that when a soft layer grows on a substrate, at the first step and near the critical threshold the structure develops surface creases, while at a higher growth ratio and far from the threshold the structure develops interfacial creases while deepening surface creases [43]. Another experimental work backed by 3D numerical simulations for a multilayer cylindrical tube mimicking growth and villi formation of the gut also showed that growth in a constrained condition triggers instability and leads to the formation of different folding patterns [8]. A study to mimic the avascular development of thin solid tumors also showed that in a circular bilayer structure formed by the growth of the outer layer (ring) on a supporting core different kinds of instabilities and patterns can be observed based on the stiffness ratio of the core to ring and thickness of the ring. With a high thickness of the growing ring and under special conditions interfacial wrinkles also were detected in the model [44].

Looking again at the bovine esophagi shown in Fig. 1, a muscle layer is much stiffer than the submucosal layer; after growth this causes interfacial creases that are detected on the interface towards the muscle layer. As predicted and modeled, interfacial creases are usually growing into the stiffer materials. These findings show that mechanical parameters play a critical role in controlling surface patterns, although the development of these patterns is believed to be the consequence of the integrated and complex interactions among genetic, biochemical, and biological processes.
IV. CONCLUSIONS

This paper explores surface and interfacial crease formation in a bilayer tubular biological tissue from an integrated theoretical and computational viewpoint. Both the stress distribution and the critical growth ratio for instability of the model and the formation of surface and interfacial creases are determined. Results show that the initial geometry, growth ratio, and shear modulus ratio of the layers are the most determinant factors for tuning the starting point and patterns of surface or interfacial creases. Usually, a growing bilayer structure prefers to develop surface creases before interfacial creases. However, in some special cases, for example, with a very thin outer layer, interfacial creases can be observed to develop before surface creases. These findings can provide some fundamental understanding in the growth modeling of tubular biological tissues such as esophagi and airways, therefore offering useful insights into the diagnosis and prevention of pathological conditions.

No research, however, provides a perfect study, with this work being no exception to the rule. More endeavors should be devoted to expanding the realm of other types of instability in the tubular structure. For example, sometimes a delamination phenomenon can be observed in layered biological tissues in the same manner as soft actuators or composite materials [45]. Also, besides biological soft tissues, the analytical and computational method presented here may be used as a tool for the quantification and calibration of soft multilayer artificial structures such as soft actuators in different environment conditions before and after the initiation of instability [46,47]. More endeavors should be devoted to exploring other various self-organized surface patterns in soft materials; these studies may open windows towards advanced functional structure as an emerging technology [48], thereby leading to a simple but fundamental platform to design and measure the different surface instabilities in soft materials.

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