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Growth and residual stresses of arterial walls

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HIGHLIGHTS

- We extend the finite deformation hyper-elastic model to study the mechanical property of the arterial wall with growth.
- We investigate the effect of growth on the deformation and the stress distribution of arterial wall.
- Analysis of axial, radial and circumferential growth is considered.
- It is shown that growth can have both stiffening and weakening effects.
- The distribution of the residual stress due to growth is given.

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ABSTRACT

Growth, residual stresses and mechanical responses of arterial walls under the inner pressure are investigated within the framework of a finite deformation hyper-elasticity theory. A biomechanical model for a two-layer thick-walled circular cylindrical tube is proposed to address the mechanical effects of finite volumetric growth and residual stresses of arterial walls. The active stress due to smooth muscle tone in the media and the dispersion of collagen fiber orientations in the adventitia are also considered. The fields of displacements and stress distributions of arterial walls with growth are solved analytically. Analysis of axial, radial and circumferential growth is considered and residual stress distributions of arterial walls in different cases of growth are compared.

1. Introduction

Vascular diseases already have become one of the leading diseases to cause significant morbidity and mortality in the world (Humphrey, 2002a, 2002b; Vorp, 2007). It has been generally accepted that mechanics plays an essential role in the process involved in vascular development and the related diseases (Holzapfel and Ogden, 2009; Taylor and Humphrey, 2009). So it is vital to understand well the mechanical properties of arterial walls. However, the mechanical behaviors of arterial walls are very complex, which are anisotropic and highly nonlinear (Taber, 2004). Pseudo-elasticity allows the use of nonlinear elasticity theories as a first approximation and this approach has provided useful approximate results in a large number of problems (Fung, 1990; Fu and Ogden, 2001; Holzapfel and Ogden, 2010a). Thus the constitutive modeling of arterial walls is the focus of researches on vascular wall mechanics (Humphrey, 2002a, 2002b; Vito and Dixon, 2003; Holzapfel and Ogden, 2003, 2006, 2009).

The macroscopic mechanical properties of materials depend on their microstructures, which results in different constitutive relations. Therefore, there is an urgent need to understand well the microstructure of arterial walls (Humphrey, 2002a, 2002b; Holzapfel et al., 2004; Gundiah et al., 2009; Watton et al., 2009; Schmid et al., 2013). From histology it is clear that an arterial wall is a multilayered structure composed of three layers with specific histological features: intima, media and adventitia. The contribution of the intima to the mechanical properties of a young arterial wall is negligible. In the media and adventitia, the elastic and collagen fibers are arranged in helically distributed families with the preferred directions (Holzapfel et al., 2000; Driessen et al., 2004).

The strain energy function proposed by Holzapfel et al. (2000), in which the collagen fibers are assumed to be perfectly aligned, works well for the media, but it does not reflect the adventitia very well (Holzapfel and Ogden, 2010a). The collagen fibers in the media are found to be arranged in two helically distributed families with a small pitch and very little dispersion in their orientation, while that in the adventitia is dispersed (Gasser et al., 2006; Ren, 2012; Schriefl

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et al., 2012). As a result, some generalized models of Holzapfel et al. (2000) considering orientation dispersions have been developed in the last years (Holzapfel et al., 2005; Gasser et al., 2006).

It is obvious that there are residual stresses in an arterial wall since it will spring open if it is sliced in the radial direction (Fung, 1990), so their effects on stress distribution should be examined (Holzapfel et al., 2000, 2007). Experiments found that the media and the adventitia have different opening angles, which indicates they should be considered in computing (Holzapfel and Gasser, 2007; Alastrue et al., 2009; Bustamante and Holzapfel, 2010; Holzapfel and Ogden, 2010b). Furthermore, when the smooth muscle orientated nearly circumferentially in the media is activated, it will alter the circumferential mechanical properties (Zulliger et al., 2004; Hayashi and Naiki, 2009). So the active stress due to smooth muscle tone in the media should be also incorporated.

In the last decade, much attention has been focused on a noteworthy property of blood vessels, their ability to grow (Dervaux and Amar, 2008; Menzel and Kuhl, 2012). Volumetric growth, the increase of bulk mass in a given system, is often involved in many fundamental biological processes such as morphogenesis, remodeling and pathological disorders (Goriely and Amar, 2005; Ambrosi et al., 2011). The artery grows in response to the rising blood pressure or the wall stress (Taber, 2001; Goriely and Vandiver, 2010; Baek and Humphrey, 2010). Growth can play a key role in the stability of a biological structure either through changes in geometry which enable the structure to better endure pressure or by creating residual stresses (Moulton and Goriely, 2011). Typical examples are mucosal folding phenomena induced by growth in esophagus, pulmonary airways, gastrointestinal tracts, blood vessels and arteries, and a sufficiently large residual stress due to growth can buckle the biological tissues (Li et al., 2011a, 2011b; Moulton and Goriely, 2011; Karsaj and Humphrey, 2012).

There is a strong coupling between mechanics and growth which is linked by residual stresses (Taber and Humphrey, 2009). In general, growth is a process of enormous complexity involving genetic, biochemical and physical components (Goriely and Amar, 2005). So there is a need to understand better the relationship between the distribution of residual stresses due to growth and the mechanical properties of arterial walls. Among three kinds of theoretical models for the volumetric growth, an elastic model based on multiplicative decomposition of deformation gradient is adopted to study the mechanical behaviors of arteries (Ambrosi et al., 2011). It is found that growth or resorption can have both stiffening and weakening effects, both stabilizing and destabilizing effects (Li et al., 2011a, 2011b; Moulton and Goriely, 2011).

In terms of growth in arterial wall, three general categories of models can be found in the literatures: volumetric growth approach, global growth approach and constrained mixture approach (Tsamis et al., 2009). Key to the volumetric growth approach is the multiplicative decomposition of the deformation gradient into an elastic and a growth part within the context of nonlinear mechanics (Rodriguez et al., 1994). This category of models was first introduced by Skalak et al. in 1982 (Skalak et al., 1982) and further developed by Rodriguez et al. (Rodriguez et al., 1994) and provided good prediction of residual stress (Rodriguez et al., 1994; Taber and Humphrey, 2009; Rachev and Gleason, 2011). Global growth approach has been proposed which consider the dependence of constituent mass fractions on wall stress, and also associated with changes in the mass fractions of the wall constituents (Schmid et al., 2009; Tsamis and Stergiopulos, 2009; Tsamis et al., 2009). Constrained mixture approach is based on the theory of constrained mixtures to track the production and removal of individual constituents (Humphrey and Rajagopal, 2002; Valentin et al., 2013). This category of models is micro-structurally motivated models to model the growth and remodeling of arteries. Other models have been proposed to consider the mechanical influence of various arterial wall constituents and assume transmurally homogeneous distributions of wall constituents (Watton et al., 2004; Watton and Hill, 2007; Schmid et al., 2009). Some recent experimental findings have shown the transmural heterogeneous constituent distribution (Kim et al., 2009).

The purpose of the present paper is to investigate the mechanical effects of growth on the mechanical response of arterial walls within the framework of finite deformation hyper-elasticity theory. Particular interest is given to residual stresses resulted from growth. The strain energy function used in the paper is the generalization model of Holzapfel et al. (2000) which has been extended recently by Gasser et al. (2006) to consider the dispersion of the collagen fiber orientations. The total deformation of the artery is decomposed into a growth part describing the addition of material and an elastic part to ensure geometric compatibility and structural integrity. Modeling the arterial wall as a two-layer thick-walled circular cylindrical tube and taking account of different residual stresses for the media and the adventitia, mechanical responses of arterial walls under the inner inflation are investigated. The fields of displacements and stress distributions are solved analytically. As isotropic growth has been well studied in the literatures, different cases of growth such as axial, radial and circumferential growth as well as isotropic growth is considered in this paper. Residual deformations and distributions of residual stresses of arterial walls in different cases of growth are given. The effects of growth are discussed in detail.

2. Strain energy functions

For a typical architecture of arterial walls, the collagen fibers are arranged in mainly two helically distributed families in their orientation (Holzapfel et al., 2000). Thus the arterial wall may be modeled as a two-family fiber-reinforced incompressible composite (Holzapfel et al., 2000; Baek et al., 2007; Gasser et al., 2006; Masson et al., 2007). The strain energy function for the media is

$$W = W_M + W_F + W_A \tag{1}$$

in which W_M is the strain energy function of the matrix and it is described by the incompressible neo-Hookean material

$$W_M = \frac{\mu^m}{2}(I_1 - 3) \tag{2}$$

in which μ^m is the shear modulus of the media. $I_1 = \tilde{\lambda}_r^2 + \tilde{\lambda}_\theta^2 + \tilde{\lambda}_z^2$ is the first invariant of the elastic deformation tensor, $\tilde{\lambda}_r$, $\tilde{\lambda}_\theta$, $\tilde{\lambda}_z$ are the principal stretches of the elastic deformation tensor. W_F is the strain energy function of the fibers and it is described by the two-fiber family model (Holzapfel et al. 2000; Gasser et al., 2006)

$$W_F = \frac{c_1}{c_2}\left\{\exp\left[c_2(I_4^* - 1)^2\right] - 1\right\} \tag{3}$$

in which the constant c_1 is the stiffness of the fibers and c_2 describes the degree of nonlinearity of the fibers. The invariant $I_4^* = kI_1 + (1-3k)I_4$, $I_4 = \mathbf{M}_\alpha(\mathbf{C}\mathbf{M}_\alpha) = \sin^2\alpha\tilde{\lambda}_\theta^2 + \cos^2\alpha\tilde{\lambda}_z^2$ is the fourth invariant of the elastic deformation tensor. $\mathbf{C}$ is the right Cauchy-Green deformation tensor, $\mathbf{M}_\alpha = [0, \sin\alpha, \cos\alpha]$ are fiber orientations in the reference configuration, and α is the angle between the direction of the fibers and the axial direction of the arterial wall. Here, assume that the symmetrical helical arrangement of the collagen fibers with their directions makes equal angles with the artery axis. k is the dispersion parameter (Gasser et al., 2006; Holzapfel and Ogden, 2010a, 2010b). W_A is the strain energy function of the smooth muscles orienting primarily in the