Constitutive framework for the modeling of damage in collagenous soft tissues with application to arterial walls

Daniel Balzani a,⁎, Sarah Brinkhues a, Gerhard A. Holzapfel b,c

a Institut für Mechanik, Fakultät für Ingenieurwissenschaften/Abteilung Bauwissenschaften, Universität Duisburg-Essen, Universitätstr. 15, 45117 Essen, Germany
b Institute of Biomechanics, Center of Biomedical Engineering, Graz University of Technology, Kronesgasse 5-I, 8010 Graz, Austria
c Institut für Mechanik, Fakultät für Ingenieurwissenschaften/Abteilung Bauwissenschaften, Universität Duisburg-Essen, Universitätstr. 15, 45117 Essen, Germany

A R T I C L E  I N F O

Article history:
Received 4 August 2011
Received in revised form 3 November 2011
Accepted 14 November 2011
Available online 3 December 2011

Keywords:
Collagenous soft tissue
Damage hysteresis
Anisotropy
Remanent strain
Arterial wall

A B S T R A C T

In this paper a new material model is proposed for the description of stress-softening observed in cyclic tension tests of collagenous soft tissues such as arterial walls, for applied loads beyond the physiological level. The modeling framework makes use of terms known from continuum damage mechanics and the concept of internal variables introducing a scalar-valued variable for the representation of fiber damage. A principle is given for the construction of damage models able to reflect remanent strains as a result of microscopic damage in the reinforcing collagen fiber families. Particular internal variables are defined able to capture the nature of arterial tissues that no damage occurs in the physiological loading domain. By application of this principle, specific models are derived and fitted to experimental data. Finally, their applicability in numerical simulations is shown by some representative examples where the damage distribution in arterial cross-sections is analyzed.

1. Introduction

Hypertension, overweight, rich alimentation, smoking, diabetes and stress may lead to biochemical and mechanical degenerative processes in collagenous soft tissues such as arterial walls. One consequence is the formation of a narrowing of the lumen, i.e. the inside space of an artery. In severe cases, when untreated, this may lead to a heart attack, a smoker’s leg or a stroke. To prevent such complications one frequently used treatment is balloon dilatation which is often accompanied by the implantation of a stent. Thereby, a balloon catheter is inserted into the affected artery and dilated with the goal to increase the lumen. During balloon inflation microscopic damage is induced in the arterial wall which also contributes to the treatment success since it results to increased strains under physiological blood pressure. In order to gain more insight into the complex biomechanical processes during therapeutic interventions such as angioplasty and for the optimization of treatment methods, the modeling of arterial tissues under supra-physiological (therapeutical) loading and related computer simulations are subject of current research.

In the last decades various experiments have been carried out to identify the material behavior of arterial walls within the physiological loading domain, see, for example, the reviews in Abé et al. [1], Fung [14], Holzapfel and Ogden [25] or Humphrey [29]. In the literature it is widely accepted that arterial walls respond quite distinctly to their mechanical environment (see, for example [31]). This poses a special challenge since healthy arteries consist of three layers with different mechanical properties: (i) the intima, the innermost layer, with a rather insignificant contribution to the solid mechanical properties for healthy young individuals. However, the intima thickens and stiffens with age (atherosclerosis) so that the mechanical contribution may become significant; (ii) the media, the middle layer, showing a complex 3D network of smooth muscle cells, elastin and collagen fibrils; (iii) the adventitia, the outermost layer, consisting of thick bundles of collagen fibrils forming a fibrous tissue. Knowledge about the mechanics of the individual layers is a prerequisite for an enhanced understanding of the complex interaction of all constituents within the arterial wall. In almost all arteries the media is stiffer than the adventitia within the low loading domain, see the experimental observations in, for example, Maltzahn et al. [32], Xie et al. [55], Yu et al. [56], or in the more recent studies for human coronary arteries [27] and human carotid arteries [48]. The above mentioned experimental approaches are related to the analysis of loading within the physiological domain. With respect to degenerative processes occurring during angioplasty especially supra-physiological loading is
required (for an overview see, for example [16]). These load levels are characterized by loading conditions much higher than those occurring under normal (physiological) conditions. Experimental studies as, for example, the one by Castaneda-Zuniga et al. [8] show that remanent deformations are obtained due to supra-physiological loading if a certain load level is exceeded. These observations are also confirmed by Oktay et al. [40] for carotid arteries of dogs, and by Holzapfel et al. [26] for human iliac arteries. Within the clinical context these effects are referred to as controlled vessel injury, see, for example, Castaneda-Zuniga [7]. The consequences of such damage are investigated in Zollikofer et al. [58] for canine arteries using electron- and optical microscopes. Profound microscopic damage is observed in the media by Castaneda-Zuniga [7]. In Schulze-Bauer et al. [46] layer-specific experiments of human adventitia are performed under physiological as well as supra-physiological conditions.

For analyzing the physiological material behavior of collagenous soft tissues numerous models have been proposed in the last decades. For example, in Vaishnav et al. [51] a two-dimensional model for a rabbit aorta is documented, while Fung et al. [15] take the exponential character of the stress–strain response into account. The proposed two-dimensional description has been extended to the three-dimensional case in Chuong and Fung [9]. In most of the recently proposed models a structural tensor is introduced with the aim to reflect the structural properties of the material leading to the invariant basis by using representation theorems for anisotropic tensor functions. The fundamental developments in the field of structural tensors are given in, for example, Boehler [5] and Spencer [50]. One of the first models for collagenous soft tissues such as tendons based on this concept was proposed by Weiss et al. [54]. However, the above mentioned models do not satisfy the polycovexity condition and thus, they do not a priori guarantee the existence of solutions of underlying boundary-value problems. In Holzapfel et al. [24] a first polyconvex model is proposed as an exponential function of the fourth mixed invariant. Further polyconvex models, which are based on the fundamental polyconvex functions for transverse isotropy and orthotropy introduced by Schröder and Neff [45], able to describe collagenous soft tissues, are proposed in, for example, Itskov and Aksel [30], Balzani [2] or Ehret and Itskov [12]. A general principle for the construction of anisotropic polyconvex functions that automatically fulfill the condition of a stress-free reference configuration and its application to arterial tissues is given in Balzani et al. [3].

Since we are mainly interested in supra-physiological loading conditions, the modeling of microscopical damage is essential. The related damage leads to a softening phenomenon of the stress–strain response and thus, increased deformations under physiological conditions. For the description of isotropic softening there exist various models. One of the first representations of damage at large strains was pursued in Simo [47], see also Govindjee and Simo [18]. Therein the authors proposed a model for polymers, which is able to describe isotropic damage in the sense of the Mullins effect. By introducing different definitions of internal variables this approach has been extended to continuous damage by Miehe [34]. Another constitutive model for the description of the Mullins effect is based on filler matrix interaction and polymer chain interaction, see Marckmann et al. [33] in order to describe damage, showing a saturation behavior during repeated unloading and reloading at fixed maximum load levels, Miehe and Keck [35] introduced a suitable model.

An alternative phenomenological form of describing damage mechanisms is linked with the notion of pseudo-elasticity. Thereby, the main idea is that different loading branches are described by different strain-energy functions. As one of the first works in this context one should mention Ogden and Roxburgh [38]. Basically, therein the Mullins effect is modeled, i.e. no remanent strains or distinct hysteresis for repeated unloading and reloading at fixed maximum load levels are obtained. An extension to the description of remanent strains is found in Ogden and Roxburgh [39], and to isotropic hysteresis in Dorfmann and Ogden [11]; specific hysteresis in partial unloading and reloading cycles are treated in Dorfmann and Ogden [10]. For the incorporation of anisotropic damage a practical approach avoiding the usage of damage tensors is given in Balzani [2], see also Schröder et al. [44], where the anisotropic damage can be described by scalar-valued variables. The recently published model by Rodríguez et al. [43] uses scalar-valued variables as well and considers a stochastic framework on the basis of the wave structure of the collagen fibers. A model for the preconditioning of soft biological tissues and the anisotropic Mullins effect is proposed in Ehret and Itskov [13]. Another recent approach provides the description of remanent strains after overstretched in the framework of finite plasticity based on the assumption of remaining deformations at the micro-scale of the fibers, see Gasser and Holzapfel [16]. A particular damage behavior for the matrix material is taken into account in, for example, Natali et al. [37] or Calvo et al. [6].

In the present study, the main goal is to define a construction principle for damage models that take into account remanent strains after unloading and to apply this principle to collagenous soft tissues such as arterial walls. Essentially, the paper is organized as follows: Section 2 explains the mathematical framework and introduces the construction principle. Furthermore, a model is given which is able to describe the complex softening hysteresis observed in cyclic uniaxial tension tests of collagenous soft tissues, where also a saturation behavior at fixed maximum load levels is shown. In Section 3 the model is specified for arterial wall tissues, and details regarding the algorithmic implementation are provided. In addition to that, it is shown that the model is able to capture the mechanical behavior of overstretched tissues also quantitatively, by fitting the model to experimental data. Section 4 provides a numerical example where the proposed model is implemented in a finite element environment, and the circumferential overstretch of a simplified atherosclerotic artery is analyzed with respect to the internal distribution of damage. Section 5 concludes the paper.

2. Mathematical modeling

2.1. Continuum mechanical framework

In the (undeformed) reference configuration $B$ the body of interest is denoted by $B \subset \mathbb{R}^3$ and parameterized in $X$; in the (deformed) current configuration it is denoted by $S \subset \mathbb{R}^3$ and parameterized in $x$. The nonlinear deformation map $\varphi : B \rightarrow S$ at time $t \in \mathbb{R}$ maps points $X \in B$ onto points $x \in S$. The deformation gradient $F$ and the right Cauchy–Green tensor $C$ are defined as

$$F(X) := \nabla \varphi(X) \quad \text{and} \quad C := F^T F$$

with the Jacobian $J := \det F > 0$. The mappings of the infinitesimal line $dX$, area $dA = N dA$ and volume elements $dV$ to their spatial counterparts $dx$, $da = nd$ and $dv$ are given by

$$dx = F dX, \quad nd = \text{Cof}[F] N dA \quad \text{and} \quad dv = J dV.$$

The cofactor is defined as $\text{Cof}[F] := J F^{-T}$. It should be mentioned that the argument $X = (F, \text{Cof}[F], \det F)$ plays an important role in the definition of polyconvexity. In the case of hyperelastic materials $W$ postulate the existence of a strain-energy function $\Psi : W \rightarrow \mathbb{R}$, defined per unit reference volume. Then we compute the second Piola–Kirchhoff stress tensor and the Cauchy stress tensor as

$$S = 2\partial_e \Psi \quad \text{and} \quad \sigma = J^{-1} F S F^T.$$
respectively. A suitable framework for the description of anisotropic materials is the concept of structural tensors, see, for example, Spencer [49], Boehler [5], or Zheng and Spencer [57]. Fiber-reinforced materials can be characterized by a given number of non-orthogonal preferred directions, then the material symmetry of the considered body is expressed by a set of second-order structural tensors

\[ M_{(a)} := A_{(a)} \otimes A_{(a)} \quad \text{with} \quad |M| = 1 \quad \text{and} \quad a = 1, \ldots, n_a, \tag{4} \]

where \( n_a \) is the number of fiber directions. For the construction of specific constitutive equations we focus on a coordinate-invariant formulation, thus, the invariants of the deformation tensor and of the structural tensors are given by

\[ I_1 := \text{tr} C, \quad I_2 := \text{det} C, \quad f_{\text{inv}} := \text{tr} [CM_{(a)}], \]

\[ J^g_5 := \text{tr} [C^T M_{(a)}], \tag{5} \]

wherein the Cofactor is defined by \( \text{Cof}[C] = \text{det} C[C]^{-1} \). Unfortunately, the fifth invariant \( f_{\text{inv}} \) is not polynomial. In order to be able to construct polyconvex strain-energy functions the alternative polyconvex invariant functions

\[ K^a_1 := \text{tr} [\text{Cof} CM_{(a)}] = f_{\text{inv}}^a - I_1 f_{\text{inv}}^g + I_2, \]

\[ K^a_2 := \text{tr} [C(I - M_{(a)})] = I_1 - f_{\text{inv}}^g, \]

\[ K^a_3 := \text{tr} [\text{Cof}(C(I - M_{(a)}))] = I_1 f_{\text{inv}}^g - f_{\text{inv}}^g \]

are introduced by Schröder and Neff [45]. Then we obtain the polynomial basis given by \( P_i := \{ I_1, I_2, I_3, f_{\text{inv}}^g, K^a_i \} \) with \( i = 1, 2, 3 \).

### 2.2. Construction of damage models for the supra-physiological domain

In the physiological loading domain, i.e. under ‘normal’ blood pressure, arteries experience an internal blood pressure of approximately up to 140 mmHg, in case of hypertension up to 180 mmHg, and sometimes even higher. Subsequently, we assume the upper limit of the physiological domain to be 180 mmHg. For the description of the physiological domain polyconvex energy functions are used. This guarantees the existence of minimizers and the Legendre–Hadamard condition is satisfied. In Balzani et al. [3], see also Balzani [2], principles for the construction of anisotropic polyconvex energies, which automatically fulfill the condition of a stress-free reference configuration, are formulated. Therefore, here we focus on functions which fit into these principles, e.g., the one proposed in Holzapfel et al. [24].

The range of loading situation beyond the physiological domain, for example, due to a balloon angioplasty or traumatic events, is referred to as supra-physiological domain. In this domain arterial tissues undergo significant softening hysteresis as a result of microscopic tissue damage. This micro-damage can be assumed as one contributor to a successful balloon angioplasty since increased strains are induced after reeding the over-expansion, see, for example, Holzapfel and Gasser [23].

#### 2.2.1. Construction principle

In order to be able to capture remanent strains by the model we formulate the construction principle.

**Consider the modified (internal) energy function**

\[ P := (1 - D)P - c \quad \text{with the damage variable} \quad D \in [0,1] \quad \text{and polyconvex function} \quad \bar{P}. \quad \text{Then include} \quad P \quad \text{into any arbitrary (external) convex and monotonically increasing function} \quad m(P(X)), \quad \text{whose first derivative is zero in the origin}. \]

The basic idea to incorporate the \((1 - D)\)-term directly into the inner function \( P \) instead of prefixing the term enables us to obtain remanent strains after unloading, which is mainly provided by the effect that the stress-free configuration is shifted by an evolution of the damage function \( \bar{D} \). Similar ideas are pursued by Govindjee and Simo [18] and Ogden and Roeburgh [39]. Possible functions \( \bar{P} \) are given by

\[ P_1 := f_{\text{inv}}^a, \quad P_2 := \bar{K}^a_1, \quad P_3 := \bar{K}^a_2, \quad P_4 := \bar{K}^a_3, \]

\[ (P_5 := I_1, \quad P_6 := I_2, \quad P_7 := I_3). \tag{7} \]

To satisfy the condition of a stress-free reference configuration for the undamaged case we subtract \( c \), which is the value of the function \( \bar{P} \) in the natural state, i.e.,

\[ c = P_i(C = I) \quad \text{for} \quad i = 1, \ldots, 7. \tag{8} \]

It should be noticed, that in case of \( D = 0 \) (hyperelastic regime) \( P \) is polyconvex and zero in the referential state, thus, it represents a special case of the construction principles given in [3,2].

Preliminary experimental investigations of cyclically overstretched arterial tissues show an additional effect: if the maximum load level is fixed in a cyclic tension test, then the stress hysteresis converges to a ‘saturated’ response curve. This behavior has to be modeled by an appropriate choice of the damage function \( D \), which is assumed to depend on the fictitiously undamaged (effective) energy \( \psi^0 = \bar{P} \), cf. [2], such that evolution of damage is activated in the loading and reloading processes. This can be achieved by defining the internal variable

\[ \beta := \langle \tilde{\mu} - \tilde{\mu}_\infty \rangle \quad \text{with} \quad \tilde{\mu} = \int_0^s \langle \psi^0(s) \rangle \, ds \tag{9} \]

with \( \psi^0 \) being the internal variable at an initial damage state in order to make sure that the damage evolution starts when entering the supra-physiological domain. The time associated to the loading history is denoted by \( s \in R^+: t \in R^+ \) defines the current loading situation. Note that the Macaulay brackets \( \langle \psi(s) \rangle = \frac{1}{2} \{ \psi(s) + |\psi(s)| \} \) filter out positive values. Then the internal variable \( \beta \) enters the damage function

\[ D(\beta) = D_s \left[ 1 - \exp \left( \frac{\ln(1 - r_s)}{\beta_s} \beta \right) \right] \quad \text{with} \quad D_s \in [0,1], \quad r_s \in [0,1], \quad \beta_s > 0 \tag{10} \]

cf. Miehe [34]. Herein, the only material parameter \( \beta_s \) is the value of the internal variable \( \beta \) which is reached at a certain fraction \( r_s \) of the maximal damage value \( D_s \) for a fixed maximum load level. We consider a fraction of \( r_s = 0.99 \) and thus, \( \beta_s \) represents the value of internal variable at a damage value which can be interpreted as saturated. The response of the damage function \( D(\beta) \) converges to a maximum value of damage \( D_s \) which is in turn not a specified number but rather a function increasing the maximally reachable damage value for increased maximum load levels. For convenience we consider the same type of function and define

\[ D(\gamma) = D_s \left[ 1 - \exp \left( \frac{\ln(1 - r_\infty)}{\gamma_\infty} \gamma \right) \right] \quad \text{with} \quad D_s \in [0,1], \quad r_\infty \in [0,1], \quad \gamma_\infty > 0, \]

where \( \gamma_\infty \) represents the value of the internal variable \( \gamma \) reached at the fraction \( r_\infty = 0.99 \) of \( D_s \) and \( D_s \) denotes a predefined converging limit for the overall damage value. In order to take into account that \( D(\gamma) \) remains unaltered for cyclic processes under fixed maximum load levels we consider the internal variable

\[ \gamma = \max_{s \in [0,b]} \left( \psi^0(s) - \psi^0_\infty \right), \tag{12} \]

which is defined as the maximum value of the effective energy reached up to the current state. Herein, \( \psi^0_\infty \) denotes the effective strain energy at an initial damage state obtained at the limit of the physiological domain. This expression leads to the saturation criterion.
\[ \psi := (\psi^0 - \psi^0_{\text{inf}}) - \gamma \leq 0. \]  
\[ \text{(13)} \]

Formally, \( D_\infty \) the maximally reachable damage value, \( \beta_0 \) the parameter for the damage behavior in the individual cycles, and \( \gamma_\infty \) the parameter describing the saturation behavior, are fitted to experimental data later on. However, since \( D_\infty \) is ideally equal to 1 and is typically set to a value close to 1 due to numerical reasons, the proposed damage model is mainly driven by the two parameters \( \beta_0 \) and \( \gamma_\infty \) that have to be determined experimentally. As can be seen in Section 3.3 the values for \( D_\infty \) vary between 0.96–0.99 and are rather close to 1.

### 2.2.2. Stresses and thermodynamical consistency

Defining the fictitiously undamaged effective energy as \( \psi^0 := \tilde{\psi} \) we focus on energy functions of the type

\[ \Psi(C, D) := m(P(C, D)) \quad \text{with} \quad P := (1 - D)\psi^0 - \epsilon. \]  
\[ \text{(14)} \]

By inserting the time derivative of the strain-energy function

\[ \ddot{\psi} - \frac{\partial \Psi}{\partial C} : \ddot{C} - \frac{\partial \Psi}{\partial D} \dot{D} + \frac{\partial \Psi}{\partial C} - \frac{m}{\partial P} \psi^0 \dot{D} \]  
\[ \text{(15)} \]

into the Clausius–Duhem inequality for isothermal conditions, where \( \dot{D} \) denotes a double contraction, we obtain

\[ \mathcal{D} = \left( \frac{1}{2} S - \frac{\partial \Psi}{\partial C} \right) : \dot{C} + m \psi^0 \dot{D} \geq 0, \]  
\[ \text{(16)} \]

with the abbreviation \( m := \partial_{\lambda} \psi^0 \). According to the standard argument of rational continuum mechanics the Clausius–Duhem inequality has to be fulfilled for each imaginable mechanical state. This leads to the equation for the second Piola–Kirchhoff stress tensor

\[ S = 2 \frac{\partial \psi^0}{\partial C} = m(1 - D)S^0 \quad \text{with} \quad S^0 := 2 \frac{\partial \psi^0}{\partial C}. \]  
\[ \text{(17)} \]

Thereby, the reduced dissipation \( \mathcal{D}_{\text{red}} \) may be provided by the inequality

\[ \mathcal{D}_{\text{red}} := m \psi^0 \dot{D} \geq 0. \]  
\[ \text{(18)} \]

Since \( m \) is assumed to be monotonically increasing and considering energy functions that are zero in the reference configuration, this means that \( \dot{D} \) has to be greater or equal zero.

By recalling (11) and (10) we obtain

\[ \dot{D}_i = -\frac{D}{\gamma_\infty} \ln(1 - r_\infty) \exp \left( \frac{\ln(1 - r_\infty)}{\gamma_\infty} \right) \geq 0 \]  
\[ \text{(19)} \]

and the evolution of the damage variable, i.e.,

\[ \dot{D} = \dot{D}_i \left[ 1 - \exp \left( \frac{\ln(1 - r_\infty)}{\beta_0} \right) \right] - \frac{D_0}{\beta_0} \ln(1 - r_\infty) \exp \left( \frac{\ln(1 - r_\infty)}{\beta_0} \right) \geq 0 \]  
\[ \text{(20)} \]

and thus, the thermodynamical consistency of damage models constructed based on the proposed construction principle is shown.

### 3. Damage model for softening hysteresis in arterial walls

Arteries are composed of (thick-walled) layers that have a qualitatively similar mechanical response. Therefore it appears to be reasonable to model each layer with the same type of strain-energy function. From the engineering point of view the layers may be considered as a composite reinforced by two families of collagen fibers. Therefore, in the sequel we concentrate on strain-energy functions of the type

\[ \Psi(C, M_1, M_2) = \psi^{\text{vol}}(\det C) + \psi^{\text{iso}}(C) + \sum_{\sigma=1}^{3} \psi_{(\sigma)}^{(i)}(C, M_{(\sigma)}). \]  
\[ \text{(21)} \]

Herein, the isotropic part \( \psi^{\text{iso}} \) represents the matrix-material (i.e., the ground substance), and \( \psi_{(\sigma)}^{(i)} \), the (transversely) isotropic energy for the fiber reinforcement in the direction \( A_{(\sigma)} \). The function \( \psi^{\text{vol}} \) is a penalty function controlling the volumetric behavior and should become large for violations of the incompressibility constraint \( J = 1 \). Due to the fact that the main load bearing elements are the fiber reinforcements the main micro-damage and, therefore, the remanent strains can be expected in particular in the fibers as well. The consideration of the main damage in the fibers has been already used by Balzani et al. [4], see also [44]. Hence, we consider the transversely isotropic part to follow the idea described in the previous section and formulate

\[ \psi_{(\sigma)}^{(i)} := m(P_{(\sigma)}(C, D_{(\sigma)}) \quad \text{with} \quad P_{(\sigma)} = (1 - D_{(\sigma)})\psi_{(\sigma)}^{(i,0)} - c \]  
\[ \text{(22)} \]

with the effective transversely isotropic strain-energy function \( \psi_{(\sigma)}^{(i,0)} = \mathcal{P}_{(\sigma)} \). The part \( (1 - D_{(\sigma)})\mathcal{P}_{(\sigma)} \) of the inner function \( P \) can be interpreted as the energy stored in the micro-fibrils, which is softened by the (scalar) damage variable \( D_{(\sigma)} \), in the case of over-expansion. The structural response and the interaction of the micro-fibrils is then captured by the (external) function \( m \). Of course, the construction principle for softening models may also be applicable to the isotropic part. An individual damage formulation for the isotropic and the anisotropic part has been proposed in, for example, Calvo et al. [6] or Peña et al. [41]. Together with proteoglycans, it is the elastin which is responsible for the resilience of the matrix [36]. Elastin is load-bearing at low and high strains, however, with a far less contribution at higher strains due to the increased bearing of load through collagen. Damage in the matrix is of secondary importance; for a detailed experimental study on human aortas see Weisbecker et al. [53]. The second Piola–Kirchhoff stress tensor is computed as

\[ S = 2 \frac{\partial \psi^0}{\partial C} = S^0 + S^{\text{iso}} = \sum_{\sigma=1}^{2} S_{(\sigma)}^{(i)}; \]  
\[ \text{(23)} \]

with the specific abbreviations

\[ S^0 = 2 \frac{\partial \psi^{\text{vol}}}{\partial C}, \quad S^{\text{iso}} = 2 \frac{\partial \psi^{\text{iso}}}{\partial C}, \quad S_{(\sigma)}^{(i)} = m'(1 - D_{(\sigma)})S_{(\sigma)}^{(i,0)}, \]  
\[ \text{(24)} \]

introduced.

### 3.1. Specification of the model

Results from the analysis by Gundiah et al. [19] show that the (classical) neo-Hookean model is a satisfactory descriptor for arterial elastin, which basically constitutes the ground-matrix from the mechanical point of view. For more details on the modeling of the mechanical response of elastin for arterial tissue see also Watton et al. [52]. Hence, the following function is considered

\[ \psi^{\text{iso}} = c_l \left( \frac{I_1}{I_0^{\text{elas}}} - 3 \right), \]  
\[ \text{(25)} \]

where \( c_l > 0 \) is a stress-like material parameter. The (strong) stiffening effect of the tissue observed at higher physiological loading (at a blood pressure of approximately 80–180 mmHg) is almost entirely due to wavy collagen fibers, which straighten with increasing deformations, see, for example, Gupta [20]. This motivates the use of an exponential function or a power function for the description of the strain energy stored in the collagen fibers. Thus, first the transversely isotropic function proposed by Holzapfel et al. [24] is combined with the fiber dispersion approach given by Gasser et al. [17], and we identify
m(P_{(a)}) = \frac{k_1}{2k_2} \left\{ \exp \left( k_2 (P_{(a)}^2) \right) - 1 \right\}, \quad P_{(a)} = \psi_{(a)}^{0,0} = \kappa I_1 + (1 - 3\kappa) f^0_j, \quad c = 1, \quad (26)

in Eq. (22). Herein, the restrictions k_1 > 0, k_2 > 0, \kappa \in [0,1/3] have to be fulfilled. The parameter k_1 > 0 is a material parameter with the dimension of stress, and k_2 > 0 is a dimensionless material parameter. An appropriate choice of k_1 and k_2 enables the assumption that the collagen fibers are almost entirely responsible for the resistance to stretch in the high loading domain (see [42]). Collagen fiber orientations in soft biological tissues such as arteries exhibit a variation in dispersion. Hence, the additional scalar parameter \kappa allows for the characterization of a state between isotropic distribution (equally distributed collagen fibers) and ideal alignment of collagen fibers. By choosing \int f^0_j for the fiber stretch measure, instead of its isotropic part, as often seen in the literature, we avoid the nonphysical response as discussed in Helfenstein et al. [21]. Finally, the complete expression for the first transversely isotropic strain-energy function, say \psi_{(a)}^{0,0HGC}, reads

\psi_{(a)}^{0,0HGC} = \frac{k_1}{2k_2} \left\{ \exp \left( k_2 \left( (1 - D_{(a)}) \left( \kappa I_1 + (1 - 3\kappa) f^0_j \right) - 1 \right)^2 \right) - 1 \right\}, \quad (27)

As mentioned above a power function appears also to be reasonable and, thus, the function proposed in [3] is again combined with the fiber dispersion approach so that we identify

m(P_{(a)}) = \alpha_1 (P_{(a)})^\alpha_2, \quad P_{(a)} = \psi_{(a)}^{0,0} = \kappa I_1 + \left( 1 - \frac{3}{2} \kappa \right) K_{j(\alpha)}, \quad c = 2, \quad (28)

with the restrictions \alpha_1 > 0, \alpha_2 > 1 (for smooth tangent moduli \alpha_2 > 2 has to be considered, \kappa \in [0,2/3]). The complete expression for the second transversely isotropic strain-energy function, say \psi_{(a)}^{0,0BNSH}, is

\psi_{(a)}^{0,0BNSH} = \alpha_1 \left( (1 - \alpha_2 D_{(a)}) \left( \kappa I_1 + \left( 1 - \frac{3}{2} \kappa \right) K_{j(\alpha)} \right)^{\alpha_2} - 2 \right). \quad (29)

Herein, the Macaulay brackets ([*]) = \frac{1}{2} ([*] + ([*])) filter out positive values. It is emphasized that the two models satisfy the construction principles in the physiological domain, as given in [3], i.e. before reaching the initial damage state, since m(P_{(a)}) is convex and monotonically increasing and P is polycrystal and zero in the reference state. Thus, the models remain polycrystal in the hyperelastic regime.

For the damage modeling we consider the model described in Section 2.2.1 and add a superscript \langle a \rangle whenever it appears to be reasonable to indicate that the associated quantities have to be evaluated for the individual fiber directions.

3.2. Algorithmic implementation

For the implementation in a finite element framework the linearization of the stresses is required. With respect to the structure of (23) the second Piola–Kirchhoff stress tensor \textbf{S} is obtained as

\Delta \textbf{S} = \frac{\partial \lambda_{vol}}{\partial \textbf{C}} : \Delta \textbf{C} + \frac{\partial \lambda_{iso}}{\partial \textbf{C}} : \Delta \textbf{C} + \sum_{\alpha=1}^{2} \left\{ \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}} : \Delta \textbf{C} + \frac{\partial \psi_{(a)}^{0,0}}{\partial D_{(a)}} \Delta D_{(a)} \right\}, \quad (30)

with the specific derivatives

\frac{\partial \psi_{(a)}^{0,0}}{\partial D_{(a)}} = -m S_{(a)}^{0,0}, \quad \Delta D_{(a)} = \frac{\partial D_{(a)}}{\partial \beta_{(a)}} \Delta \beta_{(a)} + \frac{\partial D_{(a)}}{\partial \gamma_{(a)}} \Delta \gamma_{(a)}, \quad (31)

where (24)_3, (10) and (11) have been used. By inserting the linearizations of \beta_{(a)} and \gamma_{(a)} we obtain from (31)_2

\Delta D_{(a)} = \frac{\partial D_{(a)}}{\partial \beta_{(a)}} \Delta \beta_{(a)} + \frac{\partial D_{(a)}}{\partial \gamma_{(a)}} \Delta \gamma_{(a)} = \frac{\partial \psi_{(a)}^{0,0}}{\partial \beta_{(a)}} \Delta \beta_{(a)} + \frac{\partial \psi_{(a)}^{0,0}}{\partial \gamma_{(a)}} \Delta \gamma_{(a)} = \frac{\partial \psi_{(a)}^{0,0}}{\partial \beta_{(a)}} \Delta \beta_{(a)} + \frac{\partial \psi_{(a)}^{0,0}}{\partial \gamma_{(a)}} \Delta \gamma_{(a)}, \quad (32)

Using the properties (24)_3 and \Delta \textbf{C} = 2\Delta \textbf{E} the compact notation of the incremental stress–strain relation reads

\Delta \textbf{S} = \textbf{C} : \Delta \textbf{E} \quad (33)

with the additively decoupled elasticity tensor in the material description

\textbf{C} = \textbf{C}^{elas} + \sum_{\alpha=1}^{2} \textbf{C}^{0,0}_{(a)} \textbf{C}^{0,0}_{(a)} = \textbf{C}^{elas} + \textbf{C}^{0,0}_{(a)} + \textbf{C}^{\text{iso}}_{(a)} + \textbf{C}^{\text{iso}}_{(a)} \quad (34)

Herein, the individual parts are identified as the elastic part \textbf{C}^{elas}, given as the isotropic standard elasticity tensor for hyperelasticity, i.e. [22]

\textbf{C}^{elas} = \epsilon_{vol} + \epsilon_{iso} + 4 \epsilon_{0,0} \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}}, \quad (35)

the part \textbf{C}^{0,0}_{(a)} as the (standard) damaged transversely isotropic elasticity tensor

\textbf{C}^{0,0}_{(a)} = m(1 - D_{(a)}) \textbf{C}^{0,0} + m'(1 - D_{(a)}) \textbf{S}^{0,0}_i \otimes \textbf{S}^{0,0}_i \quad (36)

with the abbreviation \textbf{m} = \partial m / \partial \psi_{(a)}^{0,0} and the effective transversely isotropic elasticity tensor

\textbf{C}^{0,0}_{(a)} = \frac{4 \epsilon_{0,0} \partial \psi_{(a)}^{0,0}}{\partial \textbf{C}}, \quad (37)

the damage evolution part

\textbf{C}^{D}_{(a)} = \begin{cases} \left[ - \textbf{m}' + m(1 - D_{(a)}) \psi_{(a)}^{0,0} \right] \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}} \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}} \otimes \textbf{S}^{0,0}_i \otimes \textbf{S}^{0,0}_i \textbf{S}^{0,0}_i & \text{if } \psi_{(a)}^{0,0} > 0, \\
0 & \text{else}, \end{cases} \quad (38)

and the saturation evolution part

\textbf{C}^{D}_{(a)} = \begin{cases} \left[ - \textbf{m}' m'(1 - D_{(a)}) \psi_{(a)}^{0,0} \right] \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}} \frac{\partial \psi_{(a)}^{0,0}}{\partial \textbf{C}} \otimes \textbf{S}^{0,0}_i \otimes \textbf{S}^{0,0}_i \textbf{S}^{0,0}_i & \text{if } \psi_{(a)}^{0,0} > 0, \\
0 & \text{else}. \end{cases} \quad (39)

For the numerical implementation we consider discrete time steps with \( t \in \{ t^{k+1}, t^k \} \) and \( k \) denoting the current and the last time step, respectively. However, we only assign the index for the current time step if necessary in order to obtain a clear notation. A trial value of the saturation criterion in the current time step can be computed by

\gamma_{(a)}^{\text{trial}} = \left( \psi_{(a)}^{0,0} - \psi_{(a)}^{0,0} \right) - k_a \gamma_{(a)}^{0,0}. \quad (40)

This value checks whether or not the Macaulay brackets in (12) becomes greater than any effective energy reached up to the current time. In the case that the value is greater than zero the algorithmic current internal variable of the saturation term is calculated as

\gamma_{(a)} = \left( \psi_{(a)}^{0,0} - \psi_{(a)}^{0,0} \right) - k_a \gamma_{(a)}^{0,0}. \quad (41)

and set equal to the value of the last time step otherwise, i.e.

\gamma_{(a)} = k_a \gamma_{(a)}^{0,0}. \quad \text{The algorithmic current internal variable of the damage term is defined as}

\beta_{(a)} = \left( \tilde{\beta}_{(a)} - \tilde{\beta}_{(a)}^{0,0} \right) \text{ with } \tilde{\beta}_{(a)} = k_a \tilde{\beta}_{(a)} + \left( \psi_{(a)}^{0,0} - k \psi_{(a)}^{0,0} \right).

(42)

The derivatives of \Delta D_{(a)} with respect to \beta_{(a)} and \gamma_{(a)} with respect to \psi_{(a)}^{0,0} follow from (10) and (42) as
\[ \frac{\partial D_{\text{vol}}}{\partial \bar{\sigma}_{\text{vol}}(\bar{\sigma}_{\text{vol}})} = -D_{\text{vol}} \ln(1 - r_1) \exp(-\ln(1 - r_1) \bar{\sigma}_{\text{vol}}) \quad (43) \]

where Eqs. (10), (11) and (41) have been used. Now we are able to compute the damage variable, the stresses and their derivatives. For an overview of the algorithm to determine the stress and elasticity tensors see Tables 1 and 2. Note that for efficiency the damage variable \( D_{\text{vol}} \) is included in the strain-energy function. Then, we focus on transversely isotropic elasticity tensors.

3.3. Method for parameter adjustment

We deduce the equations necessary for the evaluation of general anisotropic material laws for the specific boundary conditions defined by the experiments. As already mentioned, in arteries we observe mainly two fiber families oriented crosswise, thus we assume that the fibers are oriented as shown in Fig. 1.

Then the matrix notation of the associated structural tensors are computed as

\[ [M_{(1)}] = \begin{bmatrix} c^2 & -cs & 0 \\ -cs & s^2 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad \text{and} \quad [M_{(2)}] = \begin{bmatrix} c^2 & cs & 0 \\ cs & s^2 & 0 \\ 0 & 0 & 0 \end{bmatrix} \quad (46) \]

for the extension test in circumferential direction. Herein, the abbreviations \( c := \cos \beta \) and \( s := \sin \beta \) have been used. We focus on uniaxial extension tests and assume an incompressible material behavior. Therefore, the coefficients of the deformation gradient \( F \) and the right Cauchy–Green tensor \( C \) are written in terms of the stretch \( \lambda_1 \) in the direction of the extension \( x_1 \), and the transverse stretch \( \lambda_2 \) in the fiber plane, i.e.

\[
[F] = \text{diag} [\lambda_1, \lambda_2, \lambda_1^{-1} \lambda_2^{-1}] \quad \text{and} \quad [C] = \text{diag} [\lambda_1^2, \lambda_2^2, \lambda_1^{-2} \lambda_2^{-2}] \quad (47)
\]

In order to consider incompressibility the term \( \Psi^{\text{vol}} \) is included in the strain-energy function. Then, \( p \) can be interpreted as a
Fig. 1. Uniaxial extension in (a) circumferential and (b) axial direction of a specimen taken from a human blood vessel. Note that the fiber angle \( \beta_f \) describes the angle between the circumferential and the fiber direction.

Table 3
Material parameters and resulting error measure \( \bar{r} \) of the two models (45) for the media of a human carotid artery in the physiological loading domain.

<table>
<thead>
<tr>
<th></th>
<th>( c_1 [\text{kPa}] )</th>
<th>( k_1 [\text{kPa}] )</th>
<th>( k_2 [-] )</th>
<th>( z_1 [\text{kPa}] )</th>
<th>( z_2 [-] )</th>
<th>( \kappa [-] )</th>
<th>( \beta_f [-] )</th>
<th>( \bar{r} [-] )</th>
</tr>
</thead>
<tbody>
<tr>
<td>( \Psi_{(HGO)} )</td>
<td>6.56</td>
<td>1482.38</td>
<td>564.81</td>
<td>-</td>
<td>-</td>
<td>0.16</td>
<td>37.03</td>
<td>3.7 \times 10^{-2}</td>
</tr>
<tr>
<td>( \Psi_{(BNSH)} )</td>
<td>7.54</td>
<td>-</td>
<td>-</td>
<td>984.29</td>
<td>2.18</td>
<td>0.055</td>
<td>39.48</td>
<td>3.6 \times 10^{-2}</td>
</tr>
</tbody>
</table>

Fig. 2. Comparison of the constitutive model response with experimental data from uniaxial tension tests performed with strip specimens of the media of a human carotid artery in circumferential (1) and axial (2) directions. For the model response the two strain-energy functions (a) \( \Psi_{(HGO)} \) and (b) \( \Psi_{(BNSH)} \) are used.

Fig. 3. Cyclic uniaxial tension tests of the media of a human carotid artery in circumferential (1) and axial (2) directions: (a) experimental data, and (b) results of the constitutive model \( \Psi_{(HGO)} \) using the material parameters given in Tables 3 and 4.
pressure-like Lagrange multiplier. Note that neither $\Psi^{iso}$ nor $\Psi^{el}$ need necessarily to be isochoric. Following Eq. (3) we obtain the general constitutive equation

$$S = 2 \left( p \text{Cof} \left[ \epsilon \right] + \frac{\partial \Psi^{iso}}{\partial C} + \sum_{a=1}^{2} \frac{\partial \Psi^{el}}{\partial (\epsilon_{a})} \right)$$

(48)

Taking into account the symmetry of the considered problem, i.e. $\Psi^{el} := \Psi^{el}(\mathbf{2})$, and Eq. (47), we find that

$$S_{22} = 2 \left[ \frac{\partial \Psi^{iso}}{\partial \varepsilon_{12}} + \frac{\partial \Psi^{iso}}{\partial \varepsilon_{22}} \right] \left[ (\lambda_{1}^{2} + \lambda_{2}^{2} \lambda_{2})^{2} \right] + \left[ \frac{p}{C} - \frac{\partial \Psi^{iso}}{\partial \varepsilon_{12}} \right] \lambda_{2}^{2} + 2 \left[ \frac{\partial \Psi^{iso}}{\partial \varepsilon_{11}} + \frac{\partial \Psi^{iso}}{\partial \varepsilon_{22}} \left( \lambda_{1}^{2} + \lambda_{2}^{2} \lambda_{2}^{2} \right) \lambda_{2} \right]$$

(49)

in order to obtain a relation between the stretch quantities $\lambda_{1}$ and $\lambda_{2}$. It is obvious that this equation cannot be solved analytically for the general case, thus, a Newton-scheme is used. For this purpose we consider the linearization

$$\text{Lin} S_{22} = S_{22} + \frac{\partial S_{22}}{\partial \lambda_{2}} \Delta \lambda_{2} = 0,$$

(50)

where the index $(\bullet)$ denotes values at the last iteration step. Note that no index is used for the current iteration step to obtain a short notation. The iteration is repeated, where in each step the increase $\Delta \lambda_{2}$ is computed and updated until a predefined tolerance is reached ($S_{22} < \epsilon$, with $\epsilon$ close to computer accuracy). This iteration is performed for a series of $n_{\text{exp}}$ ascending experimentally measured stretches $\lambda_{2}^{\exp}$ with $j = 1, \ldots, n_{\text{exp}}$. As the starting value in each iteration procedure we set $(\lambda_{1}, \lambda_{2})$ equal to the measured extension stretch. In order to improve the efficiency of the iteration we make use of the incompressibility constraint and consider the modified starting value for

$$\lambda_{2}^{(0)} := \lambda_{2}^{(1)} \sqrt{\frac{\lambda_{1}^{(1)}}{\lambda_{1}^{(1)}}},$$

(52)

wherein the quantities $(\bullet)$ result from the iterated state of the last experimental point characterized by $\lambda_{1}^{(1)}$. With this method we are able to compute the stress $S_{11}$ in the $x_{1}$-direction governed by a general hyperelastic constitutive model as a function of the stretch $\lambda_{1}$ known from the experiment. In order to be able to compare the computed stresses with experimental data the Cauchy stresses are calculated by $\sigma^{\text{comp}} = \lambda_{1}^{2} S_{11}$. Then we define a relative error

$$r(\lambda_{1}, \mathbf{x}) := \frac{|\sigma^{\exp}(\lambda_{1}) - \sigma^{\text{comp}}(\lambda_{1}, \mathbf{x})|}{\max(\sigma^{\exp})},$$

(53)

wherein the normalization by the maximum value of experimental stresses $\max(\sigma^{\exp}) \neq 0$ in the considered extension cycle is introduced. The vector $\mathbf{x}$ contains all material parameters involved in the strain-energy function $\Psi$. For an adjustment, the resulting total error $r$, which now serves as an objective function, is given by

$$r(\mathbf{x}) = \sum_{\epsilon} \frac{1}{n_{\text{exp}}} \sum_{m=1}^{n_{\text{exp}}} \left( \frac{\sigma^{\exp}(\lambda_{1}^{(m)}) - \sigma^{\text{comp}}(\lambda_{1}^{(m)}, \mathbf{x})}{\max(\sigma^{\exp})} \right)^{2},$$

(54)

which is minimized by the use of a sequential quadratic programming algorithm. Here, we consider the number of $n_{e} = 2$ experiments: extension in circumferential and axial direction, cf. Fig. 1.

3.3.2. Adjustment results

Uniaxial tension tests, which were performed with two strip specimens from the media of a human carotid artery, were analyzed. One specimen was extended in the circumferential direction, and the other one in the axial direction, thus, the number $n_{\text{exp}}$ of experiments was two. The specimens excited during autopsy were tested within 24 h after death. For representative tissue samples see, for example, Fig. 4 in [28]. Prior to testing, pre-conditioning was achieved by executing five loading and unloading cycles for each test to obtain repeatable stress-strain curves in the physiological loading domain. Subsequently, the strips underwent uniaxial tension tests in 0.9% NaCl solution at 37 °C with continuous recording of the tensile force, the strip width and the gage length. For details on the customized tensile testing machine the reader is referred to, for example, [46]. Comprehensive biaxial data of intact and layer-dissected human carotid arteries at physiological and supra-physiological loadings are documented in [48]. First, rather moderate loads were applied in order to investigate the purest physiological domain. The number of measuring points was $n_{\text{exp}} = 49$ for the axial and $n_{\text{exp}} = 43$ for the circumferential tension test. Then the model was adjusted to the experimental data using the above mentioned method, and the resulting material parameters for the two models [45] are listed in Table 3.

The angle $\beta_{l}$ between the fiber orientation and the circumferential direction is treated as a phenomenological (fitting) parameter, which enters the structural tensors in terms of the fiber direction vectors $A_{\beta_{l}}$. Fig. 2 shows the resulting stress-strain response of the two models which is compared with experimental data. As can be seen in this figure both models lead to an accurate match of the experimental data. This is also reflected by the relatively small error measure, as given in Table 3.

Now, based on $\Psi^{(\text{HGDO})}$, we concentrate on the comparison of the model response with experimental data obtained from a significantly increased load such that the supra-physiological loading domain can be analyzed. For this purpose we analyze cyclic uniaxial tension tests performed on the specimens analyzed before. Again, we consider tests in the circumferential and the axial direction in order to obtain information with regard to the anisotropy of the tissues. The results of the experiments are shown in Fig. 3a. We observe a strong anisotropy as well as a pronounced softening hysteresis.

The material parameters of the model are adjusted such that the model response corresponds best possible to experimental data. For the least-squares fit, the (hyperelastic) parameters of the constitutive model $\Psi^{(\text{HGDO})}$, as given in Table 3, are fixed, and only the damage parameters are fitted, see Table 4 for the resulting values. Fig. 3b shows the resulting response of the proposed model $\Psi^{(\text{HGDO})}$. We observe a good qualitative and quantitative correlation with experimental data, see the error measure $r$ in Table 4. However, the model slightly overestimates the exponential character.

Alternatively, a different procedure is used for the least-squares fit. Here, the values given in Table 3 are used for the definition of suitable bounds for the (hyperelastic) parameters. Then, the hyperelastic as well as the damage parameters are fitted to the experimental data. It should be noted, that due to this procedure the hyperelastic response is not as accurately mapped as above. In addition to that, adjustment results show a low sensitivity of the overall mechanical response due to changes in $\beta_{l}$ in the range of values close to zero, i.e. for $\beta_{l} < 0.001$. Thus, the additional constraint $\beta_{l} > 0.001$ is taken into account. The obtained parameters are summarized in Table 5.
The resulting response of the models $W_{\text{HGO}}$ and $W_{\text{BNSH}}$ is shown in Fig. 4a and b, respectively. A good qualitative and quantitative correlation with the experiments is obtained for both models, see the relatively small error measure $/C^2_{22}$ provided in Table 5. However, $W_{\text{HGO}}$ slightly overestimates the exponential character, as before, whereas $W_{\text{BNSH}}$ underestimates this characteristic.

We mention here that in the matrix no microscopic damage is assumed and that the fibers are not subjected to compressive stresses. Therefore, remanent stretches after unloading are only given in the fibers for previously applied tensile stresses. Due to the superposition with the stresses given in the matrix no remanent stretches after unloading are observable in the overall response of the tissue, i.e. in the results shown in the Figs. 3 and 4.

### 4. Numerical example

In the following a numerical example is given with the goal to show the applicability of the anisotropic damage model described above. In most cases, atherosclerotic arteries with a severe stenoses (lumen reduction) are treated by a balloon angioplasty procedure in combination with the insertion of a stent. Hereby, a supra-physiological internal pressure, significantly higher than the blood pressure, is applied by inflating an inserted dilatation catheter. Hence, a circumferential overstretch of an arterial wall is numerically simulated and the distribution of damage

### Table 4

| Damage parameters and error measure $/r$ for the constitutive model $W_{\text{HGO}}$ for the media of a human carotid artery in the supra-physiological loading domain. For the adjustment the (hyperelastic) parameters are fixed, and only the damage parameters are fitted. |
|---|---|---|---|---|
| $D_\infty$ [kPa] | $\gamma_\infty$ [kPa] | $\beta_1$ [-] | $/r$ [-] |
| $W_{\text{HGO}}$ | 0.99 | 6.52 | 0.37 | 0.11 |

### Table 5

| Material parameters and error measure $/r$ of the two models (45) for the media of a human carotid artery in the supra-physiological loading domain. For the adjustment suitable bounds for the (hyperelastic) parameters, based on the results given in Table 3, have been considered. |
|---|---|---|---|---|---|---|---|---|---|---|---|---|
| $c_1$ [kPa] | $\kappa_1$ [kPa] | $\kappa_2$ [-] | $s_1$ [kPa] | $s_2$ [-] | $/D_\infty$ [kPa] | $/\gamma_\infty$ [kPa] | $\beta_1$ [-] | $/r$ [-] |
| $W_{\text{HGO}}$ | 7.50 | 1288.97 | 400.0 | - | - | 0.2 | 35.06 | 0.99 | 6.67 | 0.001 | 0.137 |
| $W_{\text{BNSH}}$ | 9.02 | - | - | 1400.0 | 2.20 | 1e-8 | 39.87 | 0.96 | 17.98 | 0.06 | 0.08 |

### Fig. 4

Cyclic uniaxial tension tests of the media of a human carotid artery in circumferential (1) and axial (2) directions: (a) results of the constitutive model $W_{\text{HGO}}$, and (b) results of the constitutive model $W_{\text{BNSH}}$. The material parameters are used from Table 5.

### Fig. 5

(a) Cross-section of the arterial model discretized with 6048 quadratic triangular finite elements (cross-section is according to [28]); the considered components are adventitia, non-diseased media, fibrotic (diseased) media, fibrous cap, lipid pool and calcification; (b) variation of the applied internal pressure $p$. 


147
computed. It is emphasized that this section provides a numerical example of limited validity with respect to quantitative results. However, this section has the goal to show that the proposed algorithm works in the context of finite element simulations. We focus on a two-dimensional simulation in order to keep the computational effort relatively low. However, for realistic simulations, in particular of diseased arteries, three-dimensional calculations are required.

A two-dimensional cross-section of an artery with a pronounced atherosclerotic plaque is considered. The components of the artery are identified by hrMRI (high resolution magnetic resonance imaging) and histological analysis ([28], see also [26]). This approach considers eight different tissue types: non-diseased intima, fibrous cap, i.e. the fibrotic part at the luminal border, fibrotic intima at the medial border, calcification, lipid pool, non-diseased media, diseased fibrotic media and adventitia. For our numerical investigations the non-diseased intima is neglected. Furthermore, the fibrotic intima at the medial border and the diseased fibrotic media are treated as one component, the fibrotic media. The cross-section including the above mentioned components is discretized with 6048 triangular elements with quadratic Ansatz functions, cf. Fig. 5a.

For the simulations the constitutive model $W_{\text{HGO}}$ is used, and for the media the parameters from Tables 3 and 4 are applied. The parameters for the adventitia are obtained by adjusting to experimental data of human carotid arteries. Due to the lack of cyclic tension tests in the supra-physiological loading range for the fibrous cap and the fibrotic media we adjust only the (hyperelastic) parameters to the experiments reported in [28], and consider the damage parameters obtained from adjusting to the media. The calcified regions are assumed to be isotropic and are modeled by setting $\psi_{\text{HGO}} = 0$. The material parameters are fitted such that the model response close to the reference configuration reflects an average Young’s Modulus of $12 \pm 4.7$ MPa, cf. [28]. The lipid pool is assumed to be a butter-like, incompressible fluid [28], which is modeled as a neo-Hookean material and due to the lack of experimental data the parameters are chosen such that a significantly lower stiffness is obtained compared with the other components. In these regions damage plays a minor role and is, therefore, neither considered in the lipid pool nor in the calcification. An overview of all parameters is listed in Table 6.

The corresponding stress–strain response of the model and the experimental data for the individual components are illustrated in Fig. 6.

![Fig. 6](image_url)

**Table 6**

<table>
<thead>
<tr>
<th>Tissue Component</th>
<th>$c_1$ [kPa]</th>
<th>$k_1$ [kPa]</th>
<th>$k_2$ [-]</th>
<th>$\kappa$ [-]</th>
<th>$\beta_1$ [-]</th>
<th>$D_\infty$ [kPa]</th>
<th>$\gamma_\infty$ [kPa]</th>
<th>$\beta_2$ [-]</th>
</tr>
</thead>
<tbody>
<tr>
<td>Adventitia</td>
<td>4.0</td>
<td>1640.23</td>
<td>115.63</td>
<td>0.097</td>
<td>45.60</td>
<td>0.99</td>
<td>10.84</td>
<td>7.36</td>
</tr>
<tr>
<td>Fibrotic media</td>
<td>21.12</td>
<td>1951.48</td>
<td>925.37</td>
<td>0.095</td>
<td>25.55</td>
<td>0.99</td>
<td>6.52</td>
<td>0.37</td>
</tr>
<tr>
<td>Fibrous cap</td>
<td>24.12</td>
<td>4778.44</td>
<td>1023.59</td>
<td>0.12</td>
<td>53.18</td>
<td>0.99</td>
<td>6.52</td>
<td>0.37</td>
</tr>
<tr>
<td>Calcification</td>
<td>2250.0</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
<tr>
<td>Lipid pool</td>
<td>2.5</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
<td>–</td>
</tr>
</tbody>
</table>

In the damage model we set $r_x = r_y = 0.99$ and in order to control the volumetric behavior the function $\Psi_{\text{HGO}} = \epsilon_1 (I_1^2 + I_2^2 - 2)$ is applied. The parameters $\epsilon_1$ and $\epsilon_2$ are chosen such that the incompressibility constraint $J = 1 \pm 1\%$ is fulfilled. For the adventitia, the non-diseased media, the fibrotic media and the fibrous...
cap the parameters are $e_1 = 50.0$ kPa and $e_2 = 20.0$; for the lipid pool we choose $e_1 = 20.0$ kPa and $e_2 = 10.0$. The calcification is not assumed to behave incompressible, and thus lower values are chosen, i.e. $e_1 = 5.0$ kPa and $e_2 = 2.0$.

Fig. 7. Distribution of the normalized damage variable $D_{(1)}/\max D_{(1)}$ in an arterial cross-section under blood pressure of 24 kPa after (a) one over-expansion (point D in Fig. 5(b)), and (b) after two over-expansions (point F in Fig. 5(b)).

Fig. 8. Distribution of the stretch $\sqrt{\frac{J_1}{4}}$ in the fiber direction of a loaded but undamaged artery at internal pressure of 24 kPa (point B in Fig. 5(b)): (a) without residual strains and (b) with axial residual strains of 5%.

Fig. 9. Distribution of the normalized damage variable $D_{(1)}/\max D_{(1)}$ in an arterial cross-section under blood pressure of 150 kPa (point E in Fig. 5(b)): (a) without and (b) with axial residual strains of 5%.
For the simulation, first an internal pressure of 24.0 kPa (±180.0 mmHg) is applied. This pressure level may be seen as an upper bound for the hypertensive pressure, and is, therefore, defined to characterize the initial damage state. In a further step the internal pressure is increased up to 150.0 kPa (±1125.0 mmHg) in order to simulate an over-expansion of the artery. Subsequently, the internal pressure is decreased until reaching a pressure of 24.0 kPa again. This situation represents the state of the artery under blood pressure after a balloon angioplasty procedure. In many cases the first overload is not sufficient for a successful balloon angioplasty treatment, and another overload has to be applied. Therefore, the over-expansion is numerically repeated as well. The complete applied loading path is depicted in Fig. 5b. In total 140 load steps are calculated until point F, where the artery is unloaded to the physiological state. For the definition of the step size an automated step control is used which repeats the load step with a smaller step size if no convergence was obtained, and which increases the step size if convergence was obtained in a very few iterations. The number of iterations for successful load steps varies between 4 and 6. The resulting distribution of the normalized damage variable, i.e. $D_{11}/\max D_{11}$, is depicted for the two situations namely where the artery is under blood pressure after a first over-expansion, see Fig. 7a (point D in Fig. 5b), and after two over-expansions, see Fig. 7b (point F in Fig. 5b). After the first expansion the damage appears mainly in the media and the fibrous cap, whereas after the second over-stretch the damage has more pronounced concentrations at the interface of the media and the fibrous cap. In addition, an increased damage is observed and higher maximum values of the normalized damage are reached.

In the previous simulation no residual strains were considered. Circumferential residual strains are associated with residual stresses which are significantly lower compared with those wall stresses obtained during an arterial over-expansion. Since the damage evolves mainly at high stress regions the negligence of circumferential residual strains may be acceptable for damage calculations. Axial residual strains, however, may have a considerable influence on the damage distribution, and for that reason a further simulation was performed where axial residual strains of 5% were prescribed. To analyze the influence of the axial residual strains on the overall biomechanical response we first compare the distribution of the fiber stretch $\left( f_{i}^{\text{F}} \right)$ in the physiological undamaged configuration at a blood pressure of 24 kPa (point B in Fig. 5b). As can be seen in Fig. 8 the stretch in the fiber direction is higher for the case where residual strains are applied (Fig. 8a), however, the difference is rather low.

In Fig. 9 the normalized damage variable $D_{11}/\max D_{11}$ is presented at a configuration where the artery is overstretched a second time (point E in Fig. 5b). Again, we compare the simulation without axial residual strains with the results obtained from applying axial residual strains of 5%. A pronounced difference between the two cases is observed, and significantly higher damage values (up to four times) are observed for the case where axial residual strains are incorporated. In addition, rather high damage values are observed at the fibrous cap – media interface, which increases the risk of plaque rupture. Note that for better comparability the same maximum damage value of $\max D_{11} = 0.0951$ has been used for the calculation of the normalized values in the Figs. 7 and 9.

5. Conclusion

The key topic in this contribution was the derivation of a principle for the construction of damage models able to describe stresssoftening hysteresis and remnant strains in the collagen fibers after unloading, which basically characterizes the supra-physiological behavior of collagenous soft tissues such as arterial walls. Using this principle, specific constitutive models have been derived and by defining suitable internal variables an undamaged physiological loading regime could be ensured. Cyclic uniaxial tension tests have been performed with the media and the adventitia of a human carotid artery, and the constructed models have been adjusted to this data. As it turned out, the models represented the experimental response with satisfactory accuracy. In order to show that the algorithmic treatment of the proposed damage model is working in finite element calculations a circumferential overstretch of a simplified atherosclerotic artery was simulated, and the resulting distribution of the damage in the arterial wall was analyzed. Although the quantitative results are not to be interpreted as realistic due to, for example, missing experimental data, a significant influence of axial residual strains on the damage distribution was identified.

Acknowledgement

The authors greatly appreciate the Deutsche Forschungsgemeinschaft (DFG) for the financial support under the research Grant BA 2823/5-1.

References
