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Computational aspects of the numerical modelling of softening, damage and permanent set in soft biological tissues

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ABSTRACT

A constitutive model is proposed for the description of the inelastic effects of biological tissues. The model can reproduce the Mullins effect, preconditioning, rupture and residual strains by the introduction of three weight factors characterizing the structural state of the material. The collagen fibers are crimped in the slack configuration and are unable to sustain load. As a result, a general theoretical and numerical framework for the constitutive modelling of biological soft tissue including inelastic effects is obtained. Finally, a comparison of numerical and experimental data is provided in order to show the reliability and effectiveness of the assumed formulation.

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1. Introduction

Numerous material models have been proposed to describe the hyperelastic behavior of fibred biological tissues in the physiological range of deformations [1–7]. All these models mentioned above have been successfully used to analyze the mechanical response of various soft tissues, as well as having offered valid alternatives to elucidate the role played by different structural aspects of the tissue in its mechanical behavior. However, most of them are limited to analyzing the mechanical response in the elastic range and not through the failure region.

Progress in experimental mechanics has significantly contributed to characterizing the mechanics of soft biological tissues. Nevertheless, difficulties persist in studying experimentally the failure behavior of fibred tissues [8–13]. These few experiments show that four important softening phenomena associated with biological tissues may be distinguished. First, the dependence of the mechanical response on the previously attained maximum load level very similar to the well-known Mullins effect in rubber-like materials [14,15]. Another typical phenomenon named preconditioning or hysteresis is characterized by continuous softening at the same load level after the first loading until reaching a certain "saturated" state [4]. Another typical phenomenon known as permanent set is characterized by residual strains after unloading [6,11,16]. Finally, the damage behavior resulting from fiber rupture and matrix disruption should be mentioned [12,17]. Fiber rupture matrix means the failure of the fibers and matrix previous to the fracture of the tissue which is not related to the Mullins effect. There are several differences between softening as a result of the structural rearrangement in the material – Mullins effect – and softening as a result of the bond rupture and complete damage.

There are several constitutive models able to describe the failure of soft tissues [18–23]; however, these models have been shown to have some drawbacks. Balzani et al. [20] assumed that discontinuous damage occurs in arterial walls and only along the fiber direction. However, other structures might also influence the failure of, for example, the vascular wall, i.e., as has been reported for the normal bovine aorta by Haslach et al. [24] or for tendons and ligaments [25]s. Thus, damage only along the fiber direction is questionable when investigating and/or describing damage of soft biological tissue. Rodriguez et al. [21,26] introduced a stochastic-structurally based damage model for fibrous soft tissues. Much simpler approaches can be used to model softening as a result of the bond rupture and complete damage as recently proposed by Volokh [22]. Only a few constitutive models have been derived to describe the loading–unloading softening behavior of soft tissues [13,26–31]. Franceschini et al. [27], Horgan and Saccomandi [32] and Weisbecker et al. [33] proposed an isotropic pseudoelastic model (the same pseudoelastic parameters for matrix and fibers) for soft tissues. Peña and Doblaré [34] present a very simple pseudo-elastic anisotropic model to reproduce the softening behavior exhibited in soft biological tissues; however,

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the pseudo-elastic model is not able to reproduce softening as a result of the bond rupture and complete damage while CDM, while other models are be able to do so. Calvo et al. [28] proposed an uncoupled directional damage model for fibred biological soft tissues that considers different damage evolutions for the matrix and for the different fiber families. In Li and Robertson [30], two damage mechanisms are coupled in a multiplicative manner. Peña et al. [31] showed that continuum damage mechanics models can reproduce the softening behavior during unloading or reloading only for low dissipative effects. On the other hand, Ehret and Itskov [29] presented a model that reproduces the softening behavior including all the tissue dissipative effects. However, the main drawback of this model is its use of non-standard invariants that leads to a very complicated approach and it is not able to reproduce the rupture process. Finally, Peña [35] presents a phenomenological model that includes the Mullins effect and rupture and residual strains of fibred biological tissues by the introduction of a new internal variable [36]. This model presents two main drawbacks. It does not include the hysteretic behavior and there is no a clear physical meaning of the parameters.

On basis of the main idea of Peña [35], this paper focuses on these latter aspects and aims to present a mathematical approach to modelling the most important aspect of softening phenomena using internal variables with physical meaning. We consider three weight factors w_i,r_k and s_k as internal variables characterizing the structural state of the material. In addition, a compact and more elegant formulation from a continuum mechanics point of view is used, the dual mechanism formulation [37,38]. Formulation and computational aspects are also discussed. The model combines simplicity and applicability, so it is a good candidate for modelling stress softening in biological soft tissues.

The paper is organized as follows. Section 2 gives a brief review of the constitutive equations for anisotropic hyperelastic materials. Section 3 describes the inelastic model for biological soft tissues. In Section 4 and 5, computational aspects related to the numerical implementation are commented on, whereas in Section 6 shows some numerical examples. Finally, Section 7 closes with some concluding remarks.

2. Previous experimental results

Inelastic phenomena which occur in mechanical testing of biological tissues are very likely to be associated with alterations in the internal structure of these materials. In particular, the alignment, state and configuration of the fibrous collagen network and the properties of the collagen fibers themselves may play a crucial role [29]. For example, Abrahams [39] suggests that strained tendons may experience "permanent structural change". He studied the histology of a tendon specimen with residual strains after unloading from uniaxial tension and indeed found that some of the collagen fibers had not returned to the original unstrained wave pattern but remained in a straightened state. In their experiments, Sellaro et al. [40] showed that collagen conformational changes and denaturation took place at an early stage of the experiments and structural changes depended significantly on the loading direction. Alastrue et al. [6] found that the residual strain, once the force reaches the zero value, depends on the maximum stretch of the sample in the previous load cycle. The residual strain is a measure of the inelastic phenomena that appears in the test, possibly plasticity and viscoelasticity of the tissue.

In order to address the above mentioned issues, and to lay the foundation for a physically motivated mechanical model for slow and large deformations of soft fibred biological tissues, in our laboratory we have performed a systematic series of in vitro experiments on animals (sheep) [6] or human tissue [16].

We summarize the tests developed in our laboratory in order to study the properties of the ovine vena cava tissue [6]. For the cyclic tests, different load and unload cycles were applied, all under force control and at the same strain rate as in the monotonic tests. Each cycle load was progressively increased 0.5 [N] and then followed by an unload cycle with a force value close to zero. The displacement rate applied to the samples was 5 [mm/min] for both samples. Three preliminary cycles at low load were applied in order to precondition the sample. Several untested and tested specimens were fixed in paraffin wax and sectioned at 4μm thickness on slides in parallel and perpendicular directions to the vessel. Hematoxylin-and-eosin staining was applied in order to observe the vena cava microstructure as well as the effects of the tests on the tissue. Due to the anisotropic nature of the tissue, both cuts in the parallel and perpendicular direction with respect to the vessel axis were studied.

A histological study both of intact and tested samples was performed. In Fig. 1(a)[1] a microphotograph of the adventitia in the direction perpendicular to the vessel axis is shown. Smooth muscle cell nuclei are stained in dark blue, whereas the muscle bundles are pink. The circular cell nuclei shape indicates that the bundles run parallel to the axial direction, constituting one of the anisotropy directions. Hematoxylin-and-eosin stains connective tissue – mainly composed of collagen and elastin – in a lighter pink, which makes it impossible to distinguish between its components. However, it can be observed that they are oriented in the view plane, that is, perpendicularly to the smooth muscle cell bundles. Fig. 1(b) shows a cut in the longitudinal direction. In this case, the main orientation of the nuclei of the smooth muscle cells matches with the microphotography vertical direction, which also matches with the vena cava longitudinal direction. Furthermore, it is noticed that the smooth muscle cell bundles interlace with the other components of the vein wall, collagen and elastin. In order to evaluate the effects of the experiments on the tissue microstructure, histological tests were performed after the tests. Fig. 1(c) depicts the histology of a tested longitudinal piece of tissue. Clear differences can be observed with respect to the intact tissue in Fig. 1(b). Accounting for the cell nuclei orientation, we conclude that testing causes permanent stretch and reorientation of the muscle cells in the test direction [41].

Two representative curves for the cyclic tests developed in this work in the circumferential and longitudinal directions are shown in Fig. 2. While not shown, it is significant than the envelope of the load-unload curves coincides closely with that of the monotonic curves [6], which corresponds to a classical Mullins effect, in which the primary loading curve is recovered after reloading from zero stress. In this type of test it was possible to study how the residual strain (λ_R), once the force reaches the zero value, depends on the maximum stretch of the sample in the previous load cycle (λ_U). The residual strain is a measure of the inelastic phenomena that appears in the test, possibly plasticity and viscoelasticity of the tissue. Even though cyclic tests showed a relatively high dispersion, a good correlation was found between λ_R and λ_U for the different samples and individuals. For circumferential cyclic tests, all data fit a quadratic polynomial ($R^2 = 0.968$), while for the longitudinal ones a linear fit is enough ($R^2 = 0.966$) (Fig. 2). This means that inelastic phenomena appear in a different manner in both directions.

With these considerations relating to the experiments developed with several biological tissues in mind, we can conclude that like the elastic properties, the inelastic behavior of soft tissues is also characterized by anisotropy. The inelastic phenomena present in the test were the Mullins effect, the preconditioning until

[1] For interpretation of color in Fig. 1, the reader is referred to the web version of this article.

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