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A constituent specific study of damage accumulation in arterial tissue

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Abstract

Introduction

Carotid atherosclerotic plaque rupture is one of the leading causes of stroke [1]. Prevention and treatments can induce tissue damage during the deployment of the intravascular device itself or through external tissue clamping during surgery.

Experimental studies have demonstrated that damage accumulation in soft tissue is associated with four phenomena; (i) the Mullins effect, (ii) hysteresis, (iii) permanent set and (iv) matrix failure and fibre rupture [2]. Although several constitutive models have been proposed to model the damage relevant phenomena in arterial tissue, there is a lack of experimental data establishing the role of the various constituents of arteries in such damage accumulation. The main aim of this research is to gain further insights into the contribution of matrix and fibres to damage accumulation in arteries under supra-physiological loading conditions.

Methods

Fresh porcine carotid arteries were dissected and cryopreserved in a freezing medium at -80°C until the time of experimentation. Cyclic mechanical tests were performed on fresh and collagenase treated dog-bone arterial samples to high strain levels.

A continuum damage mechanics (CDM) approach was employed to implement a two-constituent damage model in both matrix and fibres. Specific internal variables evolve in the model based on structural strain energy functions to capture all damage relevant phenomena in the arterial tissue.

Results

The experimental data for one representative sample and the CDM model fit are shown in Figure 1.

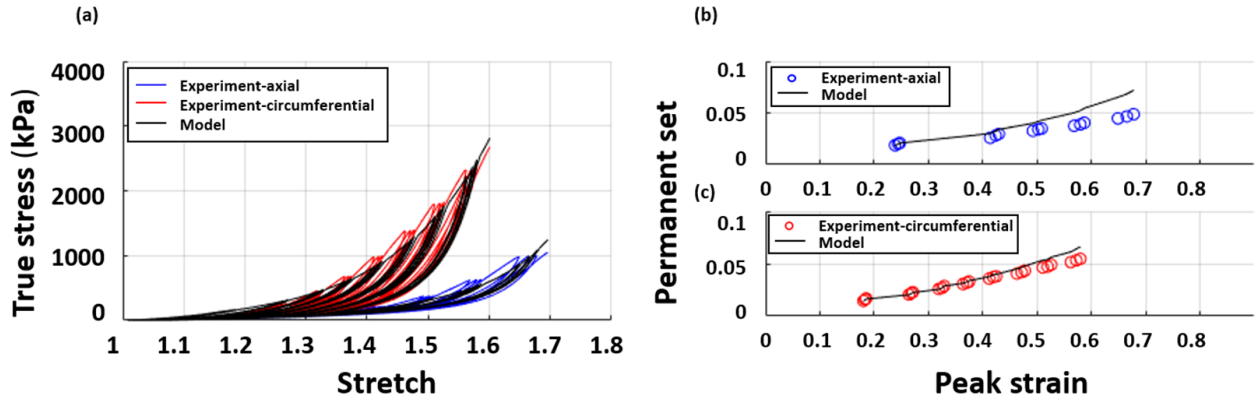

Figure 1 (a) Cyclic uniaxial tension test data for the media of a porcine carotid artery in the circumferential and axial directions and the model fitting results. Permanent set versus peak strain data in (b) axial and (c) circumferential directions.

Discussion

The experimental results show that there is no significant difference between the obtained permanent set in the intact and digested samples, or in the circumferential and axial directions for arterial tissue (Figure 1 (b) & (c)). These observations confirm the role of matrix, as opposed to fibres, in the inelastic deformation of arterial tissue at high loads. To-date, several constitutive models have assumed that damage in arterial tissue occurs only within fibres [2], and as such, those models can adequately capture the damage accumulation in the collagen fibres, but not the overall inelastic behaviour of the tissue at supra-physiological loads. The constitutive model presented here is one of the few damage models that has been established based on fitting to cyclic mechanical tests on intact and digested samples, and therefore it can elucidate the roles of softening and permanent set.

The future goal of this research is to demonstrate the influence that progressive damage accumulation has on fibre remodelling in atherosclerotic carotid artery geometries, and consequently plaque and arterial rupture.

References

[1] Mozaffarian *et al*, Circulation,133(4): e38-360, 2016

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