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DAMAGE IN COLLAGEN-RICH BIOLOGICAL TISSUES: A MULTISCALE MODELING APPROACH

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In the field of pathological processes analysis for human body, a frontier research-challenge is the mechanical modeling of biological collagen-rich tissues, which may experience non-smooth inelastic and damage mechanisms [1]. The mechanical behaviour of these tissues is strictly related with the one of their constituents (especially of collagenous bio-structures) and affects several micro (e.g., mechano-regulated tissue remodeling) and macro (e.g., mechanics of joints or vessels) physio-pathological processes. Due to the peculiar histology of collagenous tissues, characterized by a hierarchical organization from the nano (molecules), through the micro (fibers made up of bundles of cross-linked molecules, namely fibrils) up to the macroscale (tissues and organs), a multiscale approach based on tissue structure enables to analyze macroscopic effects as a consequence of altered structural features at different scales [2,3,4].

Biological tissues can experience in-vivo damage and permanent stretch due to over-physiological loads. As a matter of fact, a number of strain-controlled experiments on arterial tissue show that overstretched samples experience dissipative mechanisms leading to non-recoverable inelastic deformation before failure (i.e., residual strains) [5]. These mechanisms have to originate from inelastic effects occurring in tissue constituents, possibly regarding both non-collagenous matrix and collagen-rich bio-structures. Residual strains and damage effects in tissues are essential in explaining many physiological and pathological processes in the human organs. For instance, in a clinical context, inelastic effects occurring during angioplasty (controlled vessel injury) significantly influence both short and long-term operation success.

This work aims at gaining a deeper understanding on collagenous tissue inelastic behavior, addressing both permanent deformation and failure response. The starting point is the evidence, numerically obtained by molecular dynamical simulations, that inelastic mechanisms at the tissue macroscale are related at the fibril level to the rupture of intra-molecular and inter-molecular covalent bonds, as well as to slip-pulse mechanisms associated with inter-molecular weak interactions [6]. Therefore, the non-smooth mechanical behavior of collagenous fibrils at nano/microscale is herein modeled consistently up-scaling nanoscale molecular [7] and inter-molecular behavior by means of a multiscale homogenization technique [8]. The elasto-damage response of collagenous fibrils is modeled by means of an internal-constrained approach, introducing the fibril's free-energy and pseudo-potential of dissipation defined on the kinematic field.

Several numerical applications are discussed, aiming to highlight soundness and effectiveness of present approach, validating the modelling formulation and recovering a number of well-

established experimental evidences. Present model is open towards the possibility to correlate structure/arrangement of tissue constituents and their mechanical function, in the way of an effective integration of mechanics, biochemical surrounding and histology at different scales of investigation.

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