Effect of cigarette smoking on arterial stiffness re-interpreted using a structurally-based model

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Cigarette smoking constitutes a major risk factor for diverse cardiovascular diseases (CVD). Many physiological and pathophysiological parameters affect arterial stiffness. While underlying mechanisms remain unclear, smoking increases arterial stiffness, which contributes to many disease processes.

The goal of this work was to use a structurally motivated nonlinear constitutive relation to quantify increased arterial stiffness based on available data. Specifically, we used a “four-fiber family model” that includes dominant effects of axial, circumferential, and symmetric-diagonal families of collagen fibers embedded within an isotropic, elastin-dominated matrix. Published data, i.e. biaxial responses during pressure-diameter and axial force-length tests on pulmonary arteries from rats subjected to 2 or 3 months of smoking, were used to determine the associated best-fit values of the material parameters.

The primary finding was that cigarette smoking induces significant increases in the material parameters describing the micromechanical properties of all four families of collagen fibers with increased duration of smoking. Additionally, there was a moderate increase in the material parameter describing the behaviour of the elastic fibers. These findings suggest that arterial stiffening in response to smoking is isotropic due to the changes in the material parameters seen in all fiber directions. Although changes are manifested in both elastic and collagen fibers, the predominant stiffening appeared to be due mainly to changes in collagen fiber structure (e.g., cross-linking).