Objectives and Approaches

- Valvular interstitial cells (VICs) catabolize damaged collagen fibers and help to repair tissues. Severe collagen depletion caused by matrix metalloproteinases (MMPs) induces tissue matrix destruction, altering the viscoelastic property of the heart valve tissues.
- Collagen degradation affects cellular regulations controlled by VICs, and can lead to heart valve diseases.

Current Limitations

- It is unknown how collagen fibers are selectively catabolized or how MMPs differentiate damaged and functional collagen fibers.

Methods and Results

Stress Relaxation under Stretching and Collagen degradation

- Porcine AV and PV specimens (10mm X 10mm) are immersed in HBSS at 37°C and stretched by a biaxial tester under different strain levels.
- Two specimens are held at the assigned strain level for 10,000 seconds (about 3 hours). Collagenase replaces the HBSS at t = 3,000 for experiment tests.

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**Strain Levels and Collagenase Concentrations**

**Discussion and Conclusion**

Influence of Collagen Degradation on Mechanical Properties of Heart Valve Tissues

- The normalized stress in AV decreases as collagenase concentration increases, indicating a degradation of collagen fibers with increased simulation of MMPs (Fig.1).
- Valves tested at the physiologically accurate strain levels results in equal stress for both AV and PV (Fig. 2).
- Based on previous results [1], stretching valves at strain levels physiologically accurate for a normal heart (rather than equi-biaxial) may strengthen collagen fibers, aiding in resisting degradation from MMPs (Fig. 3).

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