DR. JUN LIAO is an Associate Professor at the University of Texas at Arlington. He received his Ph.D. in biomedical engineering from the Cleveland Clinic Foundation/Cleveland State University. After his postdoctoral training at the University of Pittsburgh, Dr. Liao has focused his research to better understand the role of biomechanics in maintaining optimal tissue performance in physiological conditions, as well as biomechanical abnormalities in diseased/injured states. The ultimate goal is to apply the gained bioengineering knowledge to innovate tissue replacement designs and medical interventions. Dr. Liao is a leading expert in tissue biomechanics and bioengineering. His productivity and contributions are evidenced by 98 peer-reviewed journal publications, 170 conference presentations/posters, 9 book chapters, and two books entitled: “Advances in Heart Valve Biomechanics” and “Advances in Biological Tissue Mechanics.” He has served as PI for AHA Grant-in-Aid and Beginning Grant-in-Aid Awards, PI for NIH R01 and R15 grants, Co-I for NIH R01, R21, R15, and T32 grants, and Co-PI for DOE and DOE grants. Due to his contributions to tissue biomechanics and bioengineering research, Dr. Liao was elected as a Fellow of the American Heart Association in 2014 and a Fellow of the American Society for Mechanical Engineers in 2020.

ABSTRACT: Collagen and elastin are major structural extracellular matrices (ECM) in heart tissues. In this talk, we introduce two mechanistic studies: (1) The role of elastin in epicardial protection; (2) collagen fibril behavior in mitral valve viscoelasticity. Study 1: During our tissue dissection from the surface of fresh porcine hearts, we discovered an interesting phenomenon: the bi-layered heart surface strip (myocardial layer + epicardial layer) always curled towards the epicardial side, revealing the existence of prestrain and residual stress in the epicardial layer. Moreover, if only the epicardial layer was dissected off the heart, the epicardial layer contracted. In other words, the epicardial layer, rich in elastin, acts like a prestrained “balloon” wrapping around a healthy heart. Our finite element analysis demonstrates that the ventricle gains additional resistance against ventricular diastolic expansion and ventricular wall protection by reducing myocardial stress, and such resistance and protection mainly derives from the prestraining instead of the epicardium alone. Study 2: To reveal collagen fibril kinematics, we developed a biaxial stretcher that fitted into synchrotron X-ray beamline. Collagen fibril D-period and the fibrillar angular distribution under stretch, creep, and stress relaxation could thus be monitored via small angle X-ray scattering. We demonstrate that collagen fibril mechanisms responsible for creep and stress relaxation in mitral valve leaflets are functionally independent, which minimalize tissue creep but allow normal stress relaxation. The obtained knowledge fills gaps in ventricular biomechanics and heart valve biomechanics and can be used to guide the design of novel biomimicking materials/bioprosthetic devices.