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Biomedical Engineering
Engineering Systems for Cardiovascular Disease Investigations

Dr. Renita Horton joined the Cullen College of Engineering at the University of Houston in January 2019. Prior to joining UH, she worked as an Assistant Professor of Agricultural and Biological engineering at Mississippi State University. Her research focuses on understanding the factors that lead to heart disease and sickle cell anemia. She is the Principal Investigator of the Cardiovascular Tissue Engineering Laboratory (CTEL) that focuses on developing tools and techniques to investigate cardiovascular disease development and progression. She received her BS in Chemical Engineering from Mississippi State University and her MS and PhD in Engineering Sciences with an enphasisn in Biomedical Engineering from Harvard University.

Abstract: Cardiovascular diseases (CVDs) are the leading cause of death globally. Central to CVDs is inflammation which contributes to tissue damage, fibrosis, and vascular and endothelial dysfunction. Underlying mechanisms remain poorly understood. Current challenges with in vitro CVD models include recapitulating the architecture of ventricular and vascular tissue, quantifying contractile function, and tissue dysfunction. Creating in vitro systems capable of mimicking both structural and functional properties of the cardiovascular system can be beneficial in mechanistic and drug efficacy studies. We utilize in vitro models inspired by in vivo vascular and myocardial properties, such as tissue mechanics, cell composition, and tissue architecture. We have utilized angiotensin II (ANG II) to elicit pathological responses in a CVD chip platform. ANG II, an octapeptide of the renin-angiotensin system, is important in cardiovascular homeostasis and pathogenesis, contributing to pathophysiological cardiac remodeling, endothelial dysfunction, and ultimately, heart failure. The goal of this study was to demonstrate that our system could effectively recapitulate features of CVD, specifically cardiac and endothelial dysfunction, by testing the effects of ANG II on the pathological remodeling of engineered cardiac and endothelial tissues. We found that ANG II exposure led to functional decline in cardiac tissues, evident in arrhythmias and decreased contractile stress generation. Further, we found that ANG II contributed to elevated reactive oxygen species levels in our

system. Results from this study can be used to design mechanistic studies to better characterize receptor contributions to physiological or pathological states and identify novel pathways for ANG II induced CVDs. We assert that this in vitro model is a suitable tool for disease studies and has the potential to serve as a testbed for drug treatment strategies.