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BIOENGINEERING

PRESENTS

From Architectural Malformations to Genetic Mutations: The Emergent Heart Function

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ABSTRACT:

The heart's primary function is to generate pressure to move blood throughout the body, which is normally accomplished with striking efficiency and is achieved by the myocardium developing force in a regular synchronized manner. Sadly, pathologies at a large range of length-scales, from molecular (ex. a single gene mutation) to tissue architecture changes (ex. hypertrophy), negatively impact the emergent force development and pumping function. Once a defect develops at any of these scales, the cells and tissues dynamically adapt, often exacerbating the pathology. However, any intervention is hindered by the lack of understanding of the mechanisms involved. In this talk, I will give two examples to illustrate how tissue engineering in combination with computational and modeling tools can be used to elucidate the emergent relationships spanning the multi-scale structure and function of the heart. In the first example, we explored how reduction of tissue architecture anisotropy reduces cardiac contractility. To accomplish this, novel parquet cardiac tissues were engineered to separate global and local architecture effects, and it was discovered that the reduction in developed force due purely to changes in global tissue architecture (>1mm) can be predicted by an astonishingly simple model, while local changes (<250 microns) trigger complex biological responses. In the second example, functional consequences of a heart disease-associated gene mutation were studied through tissue engineering of patient specific fibroblasts. The current results serve to partially unveil the mystery of why these inherited, nuclear lamina mutations result in patients presenting with symptoms of heart disease without other organ pathologies. Through both of these stories, we show how applying engineering strategies to the multi-scale intricacies of the myocardium can deepen our understanding of the heart as well as impact real patients in the future.

BIOGRAPHY:

Dr. Anna Grosberg received her undergraduate education at the University of Minnesota, double majoring in Chemical and Biomedical Engineering. Her PhD work was done at the California Institute of Technology under the guidance of Professor Mory Gharib, where she created a computational model of the myocardium mechanics. She was then a postdoctoral fellow at Harvard University in Professor Kit Parker's Disease Biophysics Group, where she worked on both computational modeling of cellular self-assembly and experimental tissue engineering device design. She started her faculty position in the Department of Biomedical Engineering in 2012, and she is a core member of The Edwards Lifesciences Center for Advanced Cardiovascular Technology. She also has a joint appointment with Department of Chemical Engineering and Materials Science and is part of the Center for Complex Biological Systems. The Grosberg lab, the Cardiovascular Modeling Laboratory, focuses on using both computational and experimental methods to investigate the structure, dynamics, and function of the heart at multiple length-scales.