THE UNIVERSITY OF ARIZONA
COLLEGE OF ENGINEERING
Biomedical Engineering

DEPARTMENT OF BIOMEDICAL ENGINEERING SEMINAR SERIES

PRESENTS

David Knoff

PhD Candidate
Biomedical Engineering
PI: Dr. Minkyu Kim
Kim Research Group

"Red Blood Cell Cytoskeleton-Inspired Biopolymer Design for Cardiovascular Biomaterials"

ABSTRACT: Natural materials can serve as great inspirational sources to develop next-generation biomaterials, attributed to their exceptional physical, chemical, and biological properties. To mimic the superior properties of natural materials, the concepts of block copolymer and polymer networks are utilized to develop well-characterized functional proteins that can be engineered into artificial protein polymers, which hierarchically assemble into nanostructured polymeric materials. In this study, we aim to mimic the reversible deformability of red blood cells to develop functional biomaterials for cardiovascular tissue engineering and drug delivery applications. Ankyrin, a red blood cell cytoskeleton protein, and streptavidin, a strong physical crosslinker, were investigated as protein building blocks for developing polymer networks with reduced topological defects and improved network homogeneity to translate single molecule protein nanomechanics to macroscale functional biomaterials.

AND

Scott Younger

PhD Student
Biomedical Engineering
PI: Dr. Fernando Teran Arce

"Medin Oligomer Membrane Pore Formation Via Non-Amyloidogenic Aggregation Pathway: Potential Mechanism of Vascular Dysfunction"

ABSTRACT: Medin, a 50 amino acid cleavage product of the lactadherin protein, is the main component of aortic medial amyloid (AMA), one of the most common forms of localized amyloid found in the vasculature of individuals older than 50 years. Previous work has implicated medin in thoracic aortic aneurysm and dissection, as well as in endothelial dysfunction related to vascular dementia and Alzheimer's disease (AD). Using lipid bilayer electrophysiology, atomic force microscopy, and thioflavin T fluorescence measurements, we found a potential oligomer-based toxicity mechanism for medin pathology: membrane pore formation. This mechanism is similar to a proposed mechanism of amyloid β (Aβ) toxicity in AD, which we have also analyzed with the same techniques. The concurrent presence of both amyloids in cerebral arterioles from AD donors, as well as their sequence similarities, suggest a potential for synergistic interactions *in vivo*.

Please join us on

Monday, November 25th, 2019

12:00-12:50 pm, Keating Bldg., Room 103

Refreshments will be available at 11:50 am

Hosts: Drs. DK Kang and Minkyu Kim

dkkang@email.arizona.edu & minkyukim@email.arizona.edu

Persons with a disability may request a reasonable accommodation by contacting the Disability Resource Center at 621-3268 (V/TTY).