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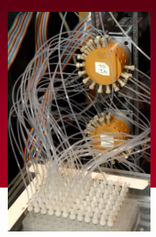
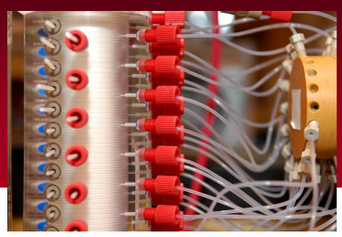



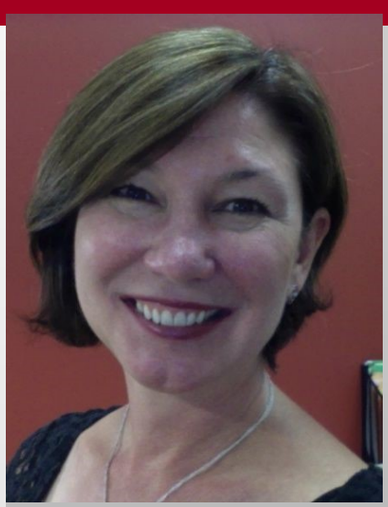

The Gene and Linda Voiland School of

Chemical Engineering and Bioengineering

2014 Seminar Series

Monday, October 27, 2014

12:00 p.m. CUE 319



Samantha Harris, Ph. D.
Associate Professor
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University of Arizona

Samantha Harris is an Associate Professor in the Department of Molecular and Cellular Medicine at the University of Arizona, in Tucson, AZ. She received her PhD from the University of Michigan in 1995 and then began training in the field of muscle physiology as a postdoc in the laboratory of Richard Moss at the University of Wisconsin. She was a faculty member in the Department of Bioengineering at the University of Washington, Seattle and then in the Department of Neurobiology, Physiology, and Behavior at the University of California, Davis prior to joining the University of Arizona in 2013. Her research interests include investigation of the molecular mechanisms by which muscle contractile proteins, especially myosin binding protein-C, regulate the force and speed of contraction and the mechanisms by which mutations in sarcomeric proteins cause hypertrophic cardiomyopathy. Her laboratory utilizes a variety of molecular, biochemical, and mechanical approaches along with development of engineered mouse models and naturally occurring large animal models of cardiac hypertrophy to study contractile protein function.

Insights into the structure and function of Cardiac Myosin Binding Protein-C.

Cardiac myosin binding protein-C (cMyBP-C) is a muscle regulatory protein necessary for normal cardiac contraction, whereas mutations in MYBPC3, the gene encoding cMyBP-C, are a prevalent cause of inherited hypertrophic cardiomyopathy (HCM) leading to heart failure in millions of people worldwide. HCM is also the most common cause of heart failure in cats and to date two causative mutations have been identified in feline MYBPC3. Here the functional and structural impact of two mutations (one human and one feline) will be discussed in the context of their 1) impact on our understanding of basic mechanisms by which cMyBP-C regulates contraction and 2) mechanisms by which mutations in cMyBP-C cause disease.