

# **BIOENGINEERING**

## **Faculty Candidate Seminar**

**Date: Friday, February 12, 2021**

**Time: 12:00 pm - 1:00pm**

**Location: Virtual**

Join Zoom Meeting:

Meeting ID: 988 0549 4005      Passcode: 454698



## **Shama Iyer, Ph.D.**

### **Biography:**

I obtained my B.S. in biological and environmental engineering and my Ph.D. in mechanical engineering from Cornell University. My dissertation research was focused on improving clinical outcomes after ventral hernia repair, by developing chitosan-coated polypropylene meshes and chitosan scaffolds for reducing fibrosis, and by promoting skeletal muscle regeneration. I am now a faculty Research Associate in the Department of Orthopaedics at the University of Maryland School of Medicine, where I study the mechanisms of progressive weakness and fragility of dystrophic skeletal muscle, using the murine (*mdx*) mouse as a model for Duchenne muscular dystrophy. My recent work examines the role of nuclear dynamics and mechanotransduction in skeletal muscle diseases. My research is currently funded by the Muscular Dystrophy Association Development Grant and the NIH K01 Mentored Research Scientist Development Award.

**Title:** Altered nucleus-cytoskeleton coupling in dystrophic muscle

### **Abstract:**

Dystrophin is a sarcolemma-associated protein, and its absence underlies the pathology in Duchenne muscular dystrophy (DMD), which is characterized by progressive muscle degeneration, weakness, and susceptibility to injury. While the genetic basis of DMD is known, the pathophysiology of muscle injury in dystrophic skeletal muscle remains unclear. The nucleus serves as the instruction manual for cells, thus how the nucleus interprets stress to alter gene expression, and its position to deliver cellular materials is paramount. This seminar will discuss the impact of aberrant nucleus-cytoskeletal coupling in *mdx* muscle (murine model of DMD) on nuclear movement and nuclear mechanotransduction. These consequent changes in nuclear movement and mechanotransduction potentially lead to gene expression differences driving the disease pathology.