BIOENGINEERING
Fall 2019 Seminar

Date: Thursday, November 7, 2019
Time: 12:00 pm - 1:00pm
Location: Exploratory Hall, Room L111
(*Videoconferencing to SciTech, K. Johnson Hall Rm 254)*

Jeremy Rotty, Ph.D.

Biography: Dr. Rotty obtained a Bachelor's degree in biology from Berea College. He then completed his PhD in the lab of Dr. Pierre Coulombe at the Johns Hopkins School of Medicine. This work revealed a novel regulatory role for keratin intermediate filaments in Src kinase signaling and cell motility, which jumpstarted his career-long interest in cell migration and the cytoskeleton. Dr. Rotty then completed his postdoctoral training in Dr. James Bear's lab at the University of North Carolina, Chapel Hill. His postdoctoral work revealed that the actin cytoskeleton is homeostatically regulated, and that the branched actin-polymerizing Arp2/3 complex is required for integrin-mediated extracellular sensing. Since 2017, Dr. Rotty has been an assistant professor in the Department of Biochemistry at the Uniformed Services University where he continues to study cell migration, cytoskeletal regulation and extracellular matrix sensing.

Title: Macrophage ECM sensing: A 'touching' story starring Arp2/3

Abstract: The branched actin-polymerizing Arp2/3 complex has recently been revealed as a critical effector of haptotaxis. Haptotaxis refers to a cell's ability to detect a gradient of substrate-bound cue (e.g. extracellular matrix) and to respond in a directional fashion by migrating 'up' the concentration gradient. Subsequent work in our lab demonstrates that Arp2/3 disruption in macrophages primes them to become hyperactivated upon inflammatory activation. Though this work reveals the branched actin cytoskeleton as a negative feedback regulator of inflammatory signaling, we are currently working to understand the mechanistic requirements for Arp2/3 in this process. A related but distinct research effort in our lab involves understanding how distinct extracellular matrix (ECM) compositions elicit strikingly different macrophage behaviors. Though it is clear that macrophages are tuned by the ECM microenvironment, we are only now beginning to understand how distinct components influence actin dynamics, myosin contractility and adhesion character. These two efforts are linked by the additional finding that distinct ECM microenvironments may influence inflammatory activation. In light of these findings, it is possible that a mechanistic link between ECM sensing and inflammatory activation will illuminate aspects of human inflammatory diseases.