Abstract:
The innate immune system acts as the body’s first line of defense against disease. Following an infection, innate immune cells are recruited to the site of the invading pathogen where they perform anti-microbial functions. This recruitment is critical for limiting the infection but must be tightly controlled by the microenvironment. Defects in leukocyte recruitment can lead to recurrent, unresolved infections and excessive leukocyte recruitment can cause chronic inflammation and tissue damage. Many different types of signals have been shown to alter leukocyte migration including extracellular matrix cues, soluble signals, cell-cell communication, and mechanical signals. Understanding how these signals contribute to leukocyte migration is important for identifying targets to control leukocyte recruitment and improve treatment options for patients. In this talk, I will discuss my research using engineered platforms to elucidate the biochemical and biophysical signals that drive leukocyte migration. I will introduce microcontact printing and traction force microscopy as methods for investigating the role of surface ligand binding and substrate stiffness on macrophage migration. Finally, I will present my newly developed, physiologically relevant, in vitro model of infection and present data showing the effect of cell-cell interactions on neutrophil recruitment to an infection.