I completed my Ph.D. training in Dr. Zamaneh Kassiri’s laboratory in the Department of Physiology in June 2018. My research focused on the role of different types of metalloproteinases in the pathogenesis of thoracic aortic aneurysm (TAA). In Dr. Kassiri’s lab, I had successfully established a murine TAA model which can recapitulate the main characteristics of human TAA. Moreover, I had also optimized isolation protocols for primary smooth muscle cells (SMC) from both mouse and human aortas, which can be used for mechanistic studies in combination with in vivo findings obtained from the murine TAA model.

Our recent paper published in *Circulation Research* is the first to identify the differential roles of SMC- and endothelial-specific disintegrin and metalloproteinase-17 (ADAM17) in the pathogenesis of experimental TAA. The protective effects of ADAM17-selective inhibitor used in wildtype mice induced with TAA further support the idea that ADAM17 can synergistically lead to TAA formation and development by promoting contractile SMCs to switch to a synthetic and pro-inflammatory state and by impairing the integrity of endothelial barrier to allow inflammatory cells to migrate into the injured site of the aorta. Taken together, our study highlighted ADAM17 as one of the major players in the pathogenesis of TAA, and inhibition of ADAM17 activity can be served as a potential therapeutic target for this life-threatening aortic disease.

Currently, I have moved to Stanford University as a postdoctoral fellow to expand my research interests to the role of vascular SMCs in other types of vascular diseases using the human pluripotent induced stem cell platform.