Mast Cells serve a vital function as the sentinels of the innate immune system. Best known for their participation in allergic reactions and inflammation, they also play an essential role in pathogen recognition and possess both antimicrobial and immunomodulatory properties. Other physiological roles have been proposed for mast cells as well such as angiogenesis and wound healing. While their role in the wound healing process remains controversial, there is evidence to suggest that they have important functions in wounds which become infected by bacteria. Leveraging mast cell function in these processes presents a potential avenue for therapeutic treatment of infected wounds which is particularly important given the growing threat of antibiotic resistant organisms. Activation using antimicrobial peptides is one potential strategy to stimulate mast cells for therapeutic purposes. Antimicrobial peptides are a class of endogenously produced molecules capable of activating immune cells as well as killing pathogens directly and have been shown to improve the healing process of both infected and uninfected wounds. Of particular interest is the poorly understood antimicrobial peptide catestatin, which has been shown to cause activation and proliferation of mast cells through an undetermined mechanism. This research aims to elucidate the mechanism by which catestatin activates mast cells, utilize a genetic mouse model of mast cell-deficiency to explore the role of mast cells in infected wounds and assess the potential for catestatin to facilitate this process.