Group A streptococcus (GAS) is a versatile human pathogen causing diseases ranging from uncomplicated mucosal infections to life-threatening invasive diseases. The development of human-relevant models of GAS infection and the introduction of new technologies have markedly accelerated the pace of discoveries related to GAS host-pathogen interactions.

By applying a signature-tagged mutagenesis and using murine model that mimics human necrotizing fasciitis (NF) we identified a novel serine protease of GAS (ScpC) which plays a central role in GAS in soft-tissue infections. Isogenic mutants deficient in ScpC constructed in two highly invasive GAS strains, M14-type isolate from a NF patient in Israel and M1T1 type, the most prevalent sterile site isolate globally, allowed us to show that ScpC is solely responsible for GAS-mediated IL-8 cleavage in-vitro, resulting in loss of its biological functions. We also demonstrated that ScpC cleaves the murine CXC chemokines, MIP-2 and KC in-vivo during the process of GAS-mediated soft-tissue infections. The cleavage of these chemokines has a major impact on host neutrophil functions. It impairs neutrophil recruitment to the site of infection, lowers neutrophil state of activation, and also reduces neutrophil extracellular trap formation, thus promoting GAS survival and proliferation.

Venue
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Admission is Free and All are Welcome