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Interactions between *Streptococcus pyogenes* and the host innate immune system that promote bacterial virulence

Approximately 250 M-protein based serotypes of Gram+ Group A *Streptococcus pyogenes* (GAS) have been isolated from infected patients. These isolates range from mild antibiotic-sensitive infections of the skin and nasopharynx to highly virulent infections of deep tissue that lead to conditions such as toxic septic shock and necrotizing fasciitis, as well as post-infective sequelae that include glomerulonephritis and rheumatic heart disease. A constant battle for survival is waged between this highly honed human-specific bacterium and the human host that can only employ generalized defense systems to combat the spread of the organism. GAS has evolved survival systems that include regulation of its own gene expression by sensors of different environmental niches, along with secretion of exotoxins that combat host defense cells and of great importance to this discussion, utilization of normal host systems for its defense. Of special interest is the conscription of the human hemostasis system by certain strains of GAS to aid this microbe in its survival and dissemination. Mechanisms will be discussed whereby components of the human coagulation, fibrinolytic, complement and inflammation systems are employed by GAS for its survival benefit.

Biography

Francis J Castellino has received his PhD degree in Biochemistry from the University of Iowa and spent two Postdoctoral years at the Duke University School of Medicine. He is the Kleiderer-Pezold Professor of Biochemistry and Director of the prestigious Keck Center for Transgene Research at the University of Notre Dame. He has published over 450 peer reviewed papers in the structural biochemistry of components of blood coagulation, anticoagulation and fibrinolysis, as well as in the pathophysiology of infective and inflammatory diseases related to hemostasis. He is a Member of several journal Editorial Boards and is the Editor-In-Chief of *Current Drug Targets*.

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