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## Francis J Castellino

University of Notre Dame, USA

Interactions of the bacterial and host systems in the persistence of group A-Streptoccal infections in humans

The virulence determinants of Gram-positive *Streptococci* are more complex than those of Gram-negative strains. For Gram-negative bacteria, lipopolysaccharide (LPS) is a primary virulence factor. In the case of the human pathogen, Gram-positive group A-*Streptococcus pyogenes* (GAS), we have examined by genomics, transcriptomics, and proteomics that several factors play various roles as virulence determinants. These include cell wall components, innate immune diversions, and other manners of avoiding the host-response. Genes acquired by GAS strains via horizontal transfer, function in virulence through acquisition of host human plasminogen which allows dissemination of the bacteria into deep tissue. The products of these genes attempt to defeat both innate and acquired immunity of the host. These virulence factors are under control of one-component and two-component bacterial regulatory systems, which regulate gene expression as needed at different stages during infection. This talk will detail the functions of bacterial virulence determinants and their dynamic interplay with the innate and acquired immune system of the host,

## **Biography**

Francis J. Castellino is the Kleiderer-Pezold Professor of Biochemistry and Director of the WM Keck Center for Transgene Research at the University of Notre Dame. This research group has studied the hemostasis system and its relation to infection and inflamation for over 40 years. He has coauthored more that 400 peer reviewed manuscripts in these areas.

fcastell@nd.edu

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