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The evolution of virulence in *Streptococcus pyogenes*

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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), several factors play various roles as virulence determinants. These include cell wall components, lipoteichoic acid and peptidoglycan, along with the cell wall capsule and exotoxins, from both the bacterial genome, e.g., the cysteine protease, streptococcal pyrogenic exotoxin (Spe)B, and from bacteriophage inserts, e.g., the DNase, streptodornase I (SdaI) and the superantigen, SpeA. Genes are also acquired by GAS strains via horizontal transfer, *e.g., emm, enn*, and *fcR* genes, as well as streptokinase (the *ska* gene), which functions in virulence through activation of host human plasminogen. 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.

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