Staphylococcus aureus proteins Sbi and Efb recruit human plasmin to degrade complement C3 and C3b.

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Projects

Complement evasion of human pathogenic microorganisms
Details

Abstract

Upon host infection, the human pathogenic microbe *Staphylococcus aureus* (*S. aureus*) immediately faces innate immune reactions such as the activated complement system. Here, a novel innate immune evasion strategy of *S. aureus* is described. The staphylococcal proteins surface immunoglobulin-binding protein (Sbi) and extracellular fibrinogen-binding protein (Efb) bind C3/C3b simultaneously with plasminogen. Bound plasminogen is converted by bacterial activator staphylokinase or by host-specific urokinase-type plasminogen activator to plasmin, which in turn leads to degradation of complement C3 and C3b. Efb and to a lesser extend Sbi enhance plasmin cleavage of C3/C3b, an effect which is explained by a conformational change in C3/C3b induced by Sbi and Efb. Furthermore, bound plasmin also degrades C3a, which exerts anaphylatoxic and antimicrobial activities. Thus, S. aureus Sbi and Efb comprise platforms to recruit plasmin(ogen) together with C3 and its activation product C3b for efficient degradation of these complement components in the local microbial environment and to protect *S. aureus* from host innate immune reactions.

Identifier

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