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Complement evasion proteins from *Staphylococcus aureus*

Understanding the mechanisms of immune evasion proteins is an important basis to develop new therapeutics against pathogenic microbes. The spread of antibiotic resistances and the high incidence of nosocomial infections caused by *Staphylococcus aureus* (*S. aureus*) illustrate the increased relevance of this facultative pathogenic bacterium as a causative agent for infections.

S. aureus uses a large panel of proteins for complement and immune evasion. Several staphylococcal immune evasion and complement regulator-binding proteins are known, like the staphylococcal binder of immunoglobulin (Sbi), the extracellular fibrinogen binding protein (Efb) and the protease staphylokinase. By using a protein microarray we identified two novel Factor H-binding proteins of S. aureus: CRASP7, that is involved in purine biosynthesis and CRASP8, which is a member of a family of superantigen-like proteins.

After identifying these immune evasion proteins aim is to characterize their mechanism of action. I am currently analyzing how these bacterial proteins influence complement activity and complement cascade progression to define the contribution of the proteins to complement evasion.

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