CspA from *Borrelia burgdorferi* inhibits the terminal complement pathway.

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Abstract

In order to survive and persist in an immunocompetent human host, Borrelia burgdorferi controls the human immune attack and blocks the damaging effects of the activated complement system. These Gram-negative spirochetes use CspA (CRASP-1) and four additional immune evasion proteins to bind combinations of human plasma regulators, including factor H, factor H-like protein 1 (FHL-1), complement factor H-related protein 1 (CFHR1), CFHR2, CFHR5, and plasminogen. As many microbial immune evasion proteins have multiple functions, we hypothesized that CspA has additional roles in complement or immune control. Here, we identify CspA as a terminal complement inhibitor. Borrelial CspA binds the human terminal complement components C7 and C9 and blocks assembly and membrane insertion of the terminal complement complex (TCC). CspA inhibits TCC assembly at the level of C7, as revealed by hemolytic assays, and inhibits polymerization of C9. CspA, when ectopically expressed on the surface of serum-sensitive Borrelia garinii, blocks TCC assembly on the level of C7 and induces serum resistance in the transformed bacteria. This CspA-mediated serum resistance and terminal complement pathway inhibition allow B. burgdorferi to survive in the hostile environment of human plasma.

Beteiligte Forschungseinheiten

Infektionsbiologie Peter F. Zipfel Mehr erfahren

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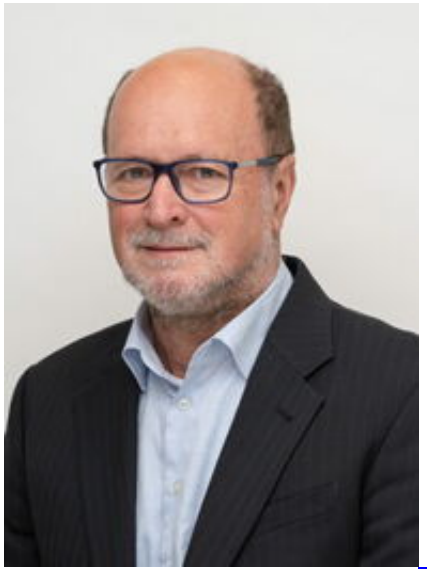

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