Contribution of the infection-associated complement regulator-acquiring surface protein 4 (ErpC) to complement resistance of *Borrelia burgdorferi*.

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Details

Abstract

Borrelia burgdorferi evades complement-mediated killing by interacting with complement regulators through distinct complement regulator-acquiring surface proteins (CRASPs). Here, we extend our analyses to the contribution of CRASP-4 in mediating complement resistance of B. burgdorferi and its interaction with human complement regulators. CRASP-4 (also known as ErpC) was immobilized onto magnetic beads and used to capture proteins from human serum. Following Western blotting, factor H (CFH), CFH-related protein 1 (CFHR1), CFHR2, and CFHR5 were identified as ligands of CRASP-4. To analyze the impact of native CRASP-4 on mediating survival of serum-sensitive cells in human serum, a B. garinii strain was generated that ectopically expresses CRASP-4. CRASP-4-producing bacteria bound CFHR1, CFHR2, and CFHR5 but not CFH. In addition, transformed spirochetes deposited significant amounts of lethal complement components on their surface and were susceptible to human serum, thus indicating that CRASP-4 plays a subordinate role in complement resistance of B. burgdorferi.

Beteiligte Forschungseinheiten

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