



**V 35 Complement regulator-acquiring surface protein 1 of *Borrelia burgdorferi* binds several ECM proteins and plasminogen**

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Lyme disease causing spirochetes *Borrelia burgdorferi*, express up to 5 complement regulator-acquiring surface proteins, termed CRASPs, which are functionally related, but structurally heterologous lipoproteins. In order to better define how CRASP-1 contributes to infection, we aimed at identifying novel human CRASP-1-binding proteins. Here, we identify plasminogen, collagen I, collagen III, collagen IV, fibronectin, and laminin as human CRASP-1-binding proteins. In order to localize the binding site of CRASP-1 for plasminogen, recombinant fragments of CRASP-1 were expressed and tested for plasminogen binding by ELISA. The plasminogen-binding regions were located to 2 separate regions of CRASP-1. As both the complement regulator factor H and plasminogen bind to CRASP-1, we analyzed whether the 2 human proteins bind simultaneously or compete for binding. Both human plasma proteins bound simultaneously to purified CRASP-1 and to intact *B. burgdorferi*. Plasminogen-bound CRASP-1 can be converted to plasmin by the urokinase-type plasminogen activator (uPA). Proteolytically active plasmin cleaves the synthetic, chromogenic substrate S-2251 and the natural substrate fibrinogen. In conclusion, CRASP-1 is a multifunctional protein of *B. burgdorferi* that binds several human extracellular matrix proteins and plasminogen. These interactions may contribute to the adhesion, bacterial colonization, and may allow dissemination of *B. burgdorferi* in the host.