Bartonella henselae trimeric autotransporter adhesin BadA expression interferes with effector translocation by the VirB/D4 type IV secretion system

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The gram-negative, zoonotic pathogen Bartonella henselae is the aetiologic agent of cat scratch disease, bacillary angiomatosis and peliosis hepatis in humans. Two pathogenicity factors of B. henselae - each displaying multiple functions in host cell interaction - have been characterized in greater detail: the trimeric autotransporter Bartonella adhesin A (BadA) and the type IV secretion system VirB/D4 (VirB/D4 T4SS). BadA mediates, e.g., binding to fibronectin (Fn), adherence to endothelial cells (ECs) and secretion of vascular endothelial growth factor (VEGF). VirB/D4 translocates several Bartonella effector proteins (Beps) into the cytoplasm of infected ECs, resulting, e.g., in uptake of bacterial aggregates via the invasome structure, inhibition of apoptosis and activation of a proangiogenic phenotype. Despite this knowledge of the individual activities of BadA or VirB/D4 it is unknown whether these major virulence factors affect each other in their specific activities. In this study, expression and function of BadA and VirB/D4 were analyzed in a variety of clinical B. henselae isolates. Data revealed that most isolates have lost expression of either BadA or VirB/D4 during in vitro passages. However, the phenotypic effects of co-expression of both virulence factors was studied in one clinical isolate that was found to stably co-express BadA and VirB/D4, as well as by ectopic expression of BadA in a strain expressing VirB/D4 but not BadA. BadA, which forms a dense layer on the bacterial surface, negatively affected VirB/D4-dependent Bep translocation and invasome formation by likely preventing close contact between the bacterial cell envelope and the host cell membrane. In contrast, BadA-dependent Fn binding, adhesion to ECs and VEGF secretion were not affected by a functional VirB/D4 T4SS. The obtained data imply that the essential virulence factors BadA and VirB/D4 are likely differentially expressed during different stages of the infection cycle of Bartonella.
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