Bartonella henselae engages inside-out and outside-in signaling by integrin β1 and talin1 during invasome-mediated bacterial uptake

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The VirB/D4 type IV secretion system (T4SS) of the bacterial pathogen Bartonella henselae (Bhe) translocates seven effector proteins (BepA-BepG) into human cells that subvert host cellular functions. Two redundant pathways dependent on BepG or the combination of BepC and BepF trigger the formation of a bacterial uptake structure termed the invasome. Invasome formation is a multi-step process consisting of bacterial adherence, effector translocation, aggregation of bacteria on the cell surface and engulfment, and eventually, complete internalization of the bacterial aggregate occurs in an F-actin-dependent manner. In the present study, we show that Bhe-triggered invasome formation depends on integrin-?1-mediated signaling cascades that enable assembly of the F-actin invasome structure. We demonstrate that Bhe interacts with integrin ?1 in a fibronectin- and VirB/D4 T4SS-independent manner and that activated integrin ?1 is essential for both effector translocation and the actin rearrangements leading to invasome formation. Furthermore, we show that talin1, but not talin2, is required for inside-out activation of integrin ?1 during invasome formation. Finally, integrin-?1-mediated outside-in signaling by FAK, Src, paxillin and vinculin is necessary for invasome formation. This is the first example of a bacterial entry process that fully exploits the bi-directional signaling capacity of integrin receptors in a talin1-specific manner.

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