A translocated protein of Bartonella henselae interferes with endocytic uptake of individual bacteria and triggers uptake of large bacterial aggregates via the invasome

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Bartonella henselae enters human endothelial cells (ECs) by two alternative routes: either by endocytosis, giving rise to Bartonella-containing vacuoles or by invasome-mediated internalization. Only the latter process depends on the type IV secretion system VirB/VirD4 and involves the formation of cell surface-associated bacterial aggregates, which get engulfed by EC membranes in an F-actin-dependent manner, eventually resulting in their complete internalization. Here, we report that among the VirB/VirD4-translocated effector proteins BepA-BepG only BepG is required for triggering invasome-mediated internalization. Expression of BepG in the Bep-deficient DeltabepA-G mutant restored invasome-mediated internalization. Likewise, ectopic expression of BepG in ECs also restored invasome-mediated internalization of the DeltabepA-G mutant, while no discernable cytoskeletal rearrangements were triggered in uninfected cells. Rather, BepG inhibited endocytic uptake of B. henselae into Bartonella-containing vacuoles and other endocytic processes, that is, invasin-mediated uptake of Yersinia enterocolitica and uptake of inert microspheres. BepG thus triggers invasome-mediated internalization primarily by inhibiting bacterial endocytosis. Bacteria accumulating on the cell surface then induce locally the F-actin rearrangements characteristic for the invasome. These cytoskeletal changes encompass both the rearrangement of pre-existing F-actin fibres and the de novo polymerization of cortical F-actin in the periphery of the invasome by Rac1/Scar1/WAVE- and Cdc42/WASP-dependent pathways that involve the recruitment of the Arp2/3 complex.
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