

## **Publication**

## Bartonella entry mechanisms into mammalian host cells

## JournalArticle (Originalarbeit in einer wissenschaftlichen Zeitschrift)

**ID** 1211772

Author(s) Eicher, S. C.; Dehio, C.

Author(s) at UniBasel Dehio, Christoph; Eicher, Simone;

**Year** 2012

Title Bartonella entry mechanisms into mammalian host cells

Journal Cellular Microbiology

Volume 14

Number 8

Pages / Article-Number 1166-73

**Mesh terms** Animals; Arthropod Vectors, microbiology; Bacterial Adhesion; Bartonella, physiology; Bartonella Infections, microbiology; Endothelium, microbiology; Erythrocytes, microbiology; Host-Pathogen Interactions; Humans

The Gram-negative genus Bartonella comprises arthropod-borne pathogens that typically infect mammals in a host-specific manner. Bartonella bacilliformis and Bartonella quintana are human-specific pathogens, while several zoonotic bartonellae specific for diverse animal hosts infect humans as an incidental host. Clinical manifestations of Bartonella infections range from mild symptoms to life-threatening disease. Following transmission by blood-sucking arthropods or traumatic contact with infected animals, bartonellae display sequential tropisms towards endothelial and possibly other nucleated cells and erythrocytes, the latter in a host-specific manner. Attachment to the extracellular matrix (ECM) and to nucleated cells is mediated by surface-exposed bacterial adhesins, in particular trimeric autotransporter adhesins (TAAs). The subsequent engulfment of the pathogen into a vacuolar structure follows a unique series of events whereby the pathogen avoids the endolysosomal compartments. For Bartonella henselae and assumingly most other species, the infection process is aided at different steps by Bartonella effector proteins (Beps). They are injected into host cells through the type IV secretion system (T4SS) VirB/D4 and subvert host cellular functions to favour pathogen uptake. Bacterial binding to erythrocytes is mediated by Trw, another T4SS, in a strictly host-specific manner, followed by pathogen-forced uptake involving the IalB invasin and subsequentreplication and persistence within a membrane-bound intraerythrocytic compartment.

Publisher American Society for Microbiology Press

**ISSN/ISBN** 1462-5814

edoc-URL http://edoc.unibas.ch/dok/A6008365

Full Text on edoc Restricted;

**Digital Object Identifier DOI** 10.1111/j.1462-5822.2012.01806.x

PubMed ID http://www.ncbi.nlm.nih.gov/pubmed/22519749

ISI-Number WOS:000306405000002

Document type (ISI) Journal Article, Review