Permissiveness of bovine epithelial cells from lung, intestine, placenta and udder for infection with *Coxiella burnetii*.

Sobotta K, Bonkowski K, Liebler-Tenorio E, Germon P, Rainard P, Hambruch N, Pfarrer C, Jacobsen ID, Menge C (2017) Permissiveness of bovine epithelial cells from lung, intestine, placenta and udder for infection with *Coxiella burnetii*. *Vet Res* 48(1), 23.

Details

PubMed

Abstract

Ruminants are the main source of human infections with the obligate intracellular bacterium Coxiella (C.) burnetii. Infected animals shed high numbers of C. burnetii by milk, feces, and birth products. In goats, shedding by the latter route coincides with C. burnetii replication in epithelial (trophoblast) cells of the placenta, which led us to hypothesize that epithelial cells are generally implicated in replication and shedding of C. burnetii. We therefore aimed at analyzing the interactions of C. burnetii with epithelial cells of the bovine host (1) at the entry site (lung epithelium) which govern host immune responses and (2) in epithelial cells of gut, udder and placenta decisive for the quantity of pathogen excretion. Epithelial cell lines [PS (udder), FKD-R 971 (small intestine), BCEC (maternal placenta), F3 (fetal placenta), BEL-26 (lung)] were inoculated with C. burnetii strains Nine Mile I (NMI) and NMII at different cultivation conditions. The cell lines exhibited different permissiveness for C. burnetii. While maintaining cell viability, udder cells allowed the highest replication rates with formation of large cell-filling Coxiella

containing vacuoles. Intestinal cells showed an enhanced susceptibility to invasion but supported C. burnetii replication only at intermediate levels. Lung and placental cells also internalized the bacteria but in strikingly smaller numbers. In any of the epithelial cells, both Coxiella strains failed to trigger a substantial IL-1β, IL-6 and TNF-α response. Epithelial cells, with mammary epithelial cells in particular, may therefore serve as a niche for C. burnetii replication in vivo without alerting the host's immune response.

Beteiligte Forschungseinheiten

Mikrobielle Immunologie Ilse Jacobsen Mehr erfahren

Leibniz-HKI-Autor*innen



Ilse Denise Jacobsen

Details

Identifier

doi: 10.1186/s13567-017-0430-9

PMID: 28403908