

Lack of potent inflammasome activation might prevent efficient *C. burnetii* clearance

Inhalt

C. burnetii is a gram-negative, obligate intracellular bacterium and the causative agent of the disease Q fever. Its primary reservoir are ruminants. Human infection can occur via the inhalation of contaminated aerosols. After infection, Q fever often is either asymptomatic or manifests as a mild flu-like illness, but pneumonia or hepatitis might also occur. In the majority of cases, the bacteria are cleared, but patients can develop chronic Q fever even years after primary infection. This indicates that in these cases the host is unable to eliminate the pathogen. An inflammatory response would be required to facilitate the clearance of the bacteria. Inflammasomes are multimeric protein complexes that induce a pro-inflammatory response to combat pathogens. Here we show that *C. burnetii* fails to cause a strong activation of the NLRP3 inflammasome, which might result in a lack of bacterial elimination. Indeed, additional stimulation with an inflammasome activator leads to inflammasome activation and, as a consequence, to reduced bacterial burden. As bacterial infections induce low oxygen levels in the affected tissue, we also investigated the impact of different oxygen concentrations on *C. burnetii* infection. Hypoxia prevents *C. burnetii* replication, without bacterial clearance. The lack of bacterial elimination might be due to the lack of inflammasome activation. Thus, forced activation of the inflammasome might be a therapeutic option for clearance of *C. burnetii*.

Keywords

NLRP3 inflammasome, inflammatory response, *C. burnetii*, hypoxia

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Professional Status of the Speaker

PhD Student

Junior Scientist Status

Yes, I am a Junior Scientist.

Thema Einordnung: Host-pathogen Interactions

Typ des Beitrags: Both Options Possible