Contribution ID: **194** Type: **Poster presentation**

Shifting towards the small cell variant form allows intracellular C. burnetii to withstand adverse microenvironmental conditions

Monday, 9 October 2023 21:06 (1 minute)

Coxiella burnetii is the causative agent of the zoonotic disease Q fever. Aside from acute fever, few patients can develop chronic Q fever months or years after primary infection, mainly characterized as endocarditis. The clinical picture of chronic Q fever suggests that *C. burnetii* establishes a persistent state. Yet, information about the induction of persistence is rare. STAT3 is important for host immunity and controls the expression of citrate transporter and citrate synthase. Under hypoxic conditions, stabilization of HIF1a impairs the STAT3 activity, resulting in reduction of the TCA cycle intermediate, citrate. Citrate limitation results in inhibition of *C. burnetii* replication without interfering with the viability of the pathogen. This suggests that under hypoxia, *C. burnetii* might undergo stringent response or enters the metabolically inactive small cell-variant form (SCV) to survive nutritional limitation. To characterize *C. burnetii* under these conditions we infected primary murine macrophages with *C. burnetii* under normoxic and hypoxic conditions and analyse the expression of stringent response and SCV genes. Our data suggests that *C. burnetii* does not undergo stringent response, but instead enters the SCV as non-replicating persistent form. Further research is required to validate this assumption. For this bacterial morphology, bacterial ability to invade new target cells and to withstand adverse conditions, e.g. drug sensitivity will be analysed.

Keywords

C. burnetii, hypoxia, small cell variant and persistence

Registration-ID code

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Professional Status of the Speaker

PhD Student

Junior Scientist Status

Yes, I am a Junior Scientist.

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Session Classification: Get-Together & Poster Viewing (P1)

Track Classification: Host-pathogen Interactions