1st Freiburg,Germany - Mendoza, Argentina Symposium on Translational Medicine

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Submitted: 23. April 2018
Published: 24. May 2018
Volume: 5
Issue: 5

Affiliation: Facultad de Ciencias Médicas Universidad Nacional de Cuyo, Argentina

Languages: English
Keywords: Cancer, Cardiovascular and Neurological Maladies
DOI: 10.17160/josha.5.5.423

Chlamydia trachomatis: PATHOGEN-HOST CELL INTERPLAY

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Chlamydia trachomatis (*Ct*) is the most prevalent sexually-transmitted bacterium worldwide. It completes its entire life cycle within human cells, inside of a modified vacuole termed inclusion. As an obligate intracellular pathogen, Ct has evolved multiple strategies to bind to, invade and parasite the host cell. In our laboratory, we aim to describe the interaction of the bacterium and the host from various approaches and scales. We have studied the manipulation of intracellular trafficking pathways executed by the bacterium to, conveniently, prevent its degradation, create a favorable niche for replication and evade the immune defense. In this way, we have reported the active recruitment of several Rab proteins and their effectors (Rab14, FIP2, Rab39a, Rab39b) to the membrane inclusion, a process that results in the acquisition of nutrients essential for growth and replication. Moreover, we study the modulation of signaling pathways (Akt, PKC) during the course of infection that may play a role in the development of Ct. To further complete the study of Ct life cycle in our team, we have described how galectin-1 enhanced its attachment to cervical epithelial cells to promote entry and invasion. A thorough understanding of the epidemiology and biology of Ct is crucial for the improvement of therapeutic strategies. For the former, we have dedicated efforts for the development of diagnostic tools of Ct and other sexually-transmitted pathogens; and for the latter, our laboratory has established a murine model of infection of Ct for *in vivo* assays of new therapeutic targets.