Lipids Droplets Play an Important Role During Obligate Intracellular Bacterial Infections

Cassandra Libbing OMS-2  
*Marian University - Indianapolis*

Rea-Mae Azcueta  
*Marian University - Indianapolis*

Minal Mulye Ph.D  
*Marian University - Indianapolis*

Follow this and additional works at: https://mushare.marian.edu/mucom_rd

Part of the Medicine and Health Sciences Commons

Recommended Citation

Libbing, Cassandra OMS-2; Azcueta, Rea-Mae; and Mulye, Minal Ph.D, "Lipids Droplets Play an Important Role During Obligate Intracellular Bacterial Infections" (2018). MU-COM Research Day. 93.  
https://mushare.marian.edu/mucom_rd/93

This Poster is brought to you for free and open access by the College of Osteopathic Medicine at MUSHare. It has been accepted for inclusion in MUCOM Research Day by an authorized administrator of MUSHare. For more information, please contact emandity@marian.edu.
Lipid droplets play an important role during obligate intracellular bacterial infections
Cassandra Libbing, Rea-Mae Azcuetla, Minal Mulve
Marian University College of Osteopathic Medicine, Indianapolis, IN

Chlamydia pneumoniae

Causative agent of: infectious, pharyngitis, bronchitis, pneumonia, and linked to atherosclerosis.

Pathogenesis:
- C. pneumoniae deposits in endoplasmic reticulum, monocytes, and endothelial cells
- Manipulates host mechanisms by inducing expression of inflammatory cytokines, pro-inflammation, and metalloproteinase, and fusogenic

Lipid Droplets and C. pneumoniae:
- C. pneumoniae is found around LDs of atherosclerotic plaques, amastigote as pathogen given

Pathogenetic host cell formation:
- Disregulates HIF-NFκB and PI3K-Akt, thus releasing inhibition of ADAP1 in macrophages and progression of macrophage foam cell of atherosclerotic lesions
- Treatment with PI3K and PIM1's activity significantly inhibits C. pneumonia induced foam cell formation.

Coxiella burnetii

Causative agent of: Louseborne and tick-borne relapsing fever and enzootic meningitis

Pathogenesis:
- Granuloma; infects macrophages
- Causes and replicates inside a parasitophorous vacuole (PV)
- Releases bacterial effectors via T4SS (Type IV secretion system) to manipulate host cell functions

Anaplasma phagocytophilum & Ehrlichia chaffeensis

Causative agent of: human granulocytic anaplasmosis and human monocytic ehrlichiosis, respectively.

Pathogenesis:
- Obligate intracellular tick-borne pathogens
- Enters cells via phagocytosis
- Enters through cell-associated endocytosis

Lipid droplets and C. burnetii:
- C. burnetii induces LD accumulation in macrophages
- Blocks LD formation increases bacterial growth
- Blocks LD breakdown promotes bacterial growth
- Suggests that LD breakdown is vital for C. burnetii survival and proliferation in in vivo macrophages

Anaplasma phagocytophilum is a pathogen of the tick-borne fever complex and a causative agent of human granulocytic anaplasmosis.

Acknowledgements
The lipid droplet dream team (Adam McDett, Ahila, Cassie Libbing, and Rea-Mae Azcuetla) and our wonderful PI, Dr. Minal Mulve.