Analysis of Chlamydia pneumoniae-infected monocytes following incubation with a novel peptide, acALY18, implicates the inflammasome in clearance of infection

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Chlamydia pneumoniae infection may be a trigger for the pathology observed in sporadic late-onset Alzheimer’s disease (1). Analysis of the C. pneumoniae genome and its transcriptome suggests that multiple pathways are involved in the infection, including innate and adaptive immune responses (2). However, the mechanisms underlying the interaction between C. pneumoniae and human host cells remain largely unknown. In this study, we investigate the role of C. pneumoniae infection in innate and adaptive immune responses in human monocytes and endothelial cells, using microarray analysis and functional assays.

**Results**

**Topography of TRPC1**

Infection of human monocytes with C. pneumoniae (Phila1 strain) results in a significant increase in the expression of several genes related to the TRPC1 channel, a key component of the innate immune response. These findings are consistent with previous studies showing that C. pneumoniae infection induces expression of TRPC1 in monocytes (3). The upregulation of TRPC1 in response to C. pneumoniae infection suggests a role for this channel in the innate immune response to this pathogen.

**Synthetic PDAG peptide (acALY18) decreases infection in monocytes**

AcALY18, a synthetic peptide derived from the lysosomal damage response, has been shown to induce NALP3 activation (4). Using a peptide such as acALY-18, which was originally derived and identified from the lysosomal damage response, we demonstrate that this peptide promotes clearance of C. pneumoniae-infected monocytes. This finding is consistent with previous studies showing that the lysosomal damage response is involved in the clearance of C. pneumoniae (5). The results of this study suggest that acALY18 may be a potential therapeutic agent for treating C. pneumoniae infection.

**Innate and adaptive immunity gene expression**

C. pneumoniae infection alters gene expression for markers of innate immunity, including toll-like receptor (TLR) ligands and inflammasome components. These findings are consistent with previous reports showing that C. pneumoniae infection induces expression of TLRs and inflammasome components in monocytes (6). The results of this study suggest that C. pneumoniae infection promotes inflammation through the activation of innate immune responses.

**Alzheimer gene expression in C. pneumoniae infected versus uninfected human THP1 monocytes**

C. pneumoniae infection induces expression of genes associated with innate and adaptive immunity, including TLRs and inflammasome components. However, C. pneumoniae infection does not induce expression of genes associated with Alzheimer’s disease, such as amyloid beta (Aβ) precursor protein (APP) and tau. These findings suggest that C. pneumoniae infection promotes inflammation through the activation of innate immune responses, but does not promote inflammation through the activation of adaptive immune responses.

**Conclusions**

- C. pneumoniae infection induces inflammation through the activation of innate immune responses.
- C. pneumoniae infection does not induce inflammation through the activation of adaptive immune responses.
- The role of C. pneumoniae infection in the development of Alzheimer’s disease remains to be fully elucidated.

The results of this study provide new insights into the mechanisms underlying the interaction between C. pneumoniae and human host cells. These findings suggest that C. pneumoniae infection promotes inflammation through the activation of innate immune responses, but does not promote inflammation through the activation of adaptive immune responses.

**References**