Chlamydia pneumoniae infection of neuronal cells induces changes in calcium-associated gene expression consistent with Alzheimer’s disease

Christopher Andrew Cappellini  
Philadelphia College of Osteopathic Medicine, christophercap@pcom.edu

Ahmad B. Cader  
Philadelphia College of Osteopathic Medicine, ahmadca@pcom.edu

Keith G. Williams  
Philadelphia College of Osteopathic Medicine, keithwi@pcom.edu

Juliana Zoga  
Philadelphia College of Osteopathic Medicine, JulianaZo@pcom.edu

Susan T. Hingley  
Philadelphia College of Osteopathic Medicine, susanh@pcom.edu

Follow this and additional works at: http://digitalcommons.pcom.edu/posters

Part of the Bacterial Infections and Mycoses Commons, Medical Cell Biology Commons, and the Nervous System Diseases Commons

Recommended Citation
Cappellini, Christopher Andrew; Cader, Ahmad B.; Williams, Keith G.; Zoga, Juliana; and Hingley, Susan T., "Chlamydia pneumoniae infection of neuronal cells induces changes in calcium-associated gene expression consistent with Alzheimer’s disease" (2013). Scholarly Posters. Book 6.  
http://digitalcommons.pcom.edu/posters/6

This Book is brought to you for free and open access by DigitalCommons@PCOM. It has been accepted for inclusion in Scholarly Posters by an authorized administrator of DigitalCommons@PCOM. For more information, please contact library@pcom.edu.
Chlamydia pneumoniae infection of neuronal cells induces changes in calcium-associated gene expression consistent with Alzheimer’s disease

Christopher A. Cappellini, Ahmad B. Cader, Keith G. Williams, Juliana D. Zoga, Susan T. Hingley, Brian J. Balin, Denah M. Appelt, Marcus G. Bell

Center for Chronic Disorders of Aging (CCDA), Philadelphia College of Osteopathic Medicine, Philadelphia, PA

Abstract

Background and Significance: Previous studies have shown that cells infected with Chlamydia pneumoniae (Cpn) exhibit altered gene expression consistent with that observed in Alzheimer’s disease (AD). Furthermore, AD neuropathogenesis has been linked to dysregulation of intracellular calcium and related processes. Therefore, we hypothesized that one mechanism by which pathogenesis evolves in AD is through infection-induced changes in calcium homeostasis.

Objectives: To determine if infection of neuronal cells with Cpn alters expression of calcium-related genes associated with neurodegeneration.

Methods: SK-N-MC neuronal cells were infected with Cpn (MOI=1; 3, 24 or 72 hours). Then, total RNA was isolated and real-time PCR analysis was performed (SABiosciences PAMHS-066).

Results: Following infection, approximately 29 genes displayed regulation changes of 2-fold or greater, including genes pertaining to neurotransmitters, cell cycle and immune regulations, and other calcium-regulating elements. Genes involved in synaptic function and memory such as AREG, ATF3, EG2, and GG1 were initially up-regulated, but fell to baseline or below 72 hours. Genes of the altered gene have been involved in AD pathogenesis.

Conclusions: Our data suggest that Cpn alters calcium-related gene expression in host neuronal cells. This work was funded by the Center for Chronic Disorders of Aging (CCDA) at the Philadelphia College of Osteopathic Medicine and the Adolph and Rose Levis Foundation for Alzheimer’s disease research.

Results

- Heat Map Analysis of 84 Calcium-Related Genes
- Gene Symbol
- Gene Name

Materials & Methods

Neuronal cells, SK-N-MC (ATCC, HTB-10), were infected with ATCC’s MOI-states of Chlamydia pneumoniae as MOI of 1 for 3,24 or 72 hours. The Signal Transduction PathwayFinder Array from Qiagen (SABiosciences) was used to analyze the expression of calcium-related genes. All experiments were performed in triplicate.

References

This work was funded by the Center for Chronic Disorders of Aging (CCDA) at the Philadelphia College of Osteopathic Medicine and the Adolph and Rose Levis Foundation for Alzheimer’s disease research.