Chlamydophila (Chlamydia) pneumoniae promotes Ab 1-42 amyloid processing in Neuronal Cells: A Pathogenic Trigger for Alzheimer's Disease

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C. pneumoniae (Chlamydia) pneumoniae promotes Aβ 1-42 amyloid processing in Neuronal Cells: A Pathogenic Trigger for Alzheimer's Disease

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Abstract

**Background:** Previous studies have identified Chlamydia pneumoniae (C. pneumoniae) infection of neuronal cells to produce increased amyloid precursor protein (APP) levels. Western blot analyses have demonstrated increased APP levels in cortex and hippocampus of AD brains. Neuronal infection with C. pneumoniae has been associated with increased cytotoxicity and neuronal death. In addition, C. pneumoniae has been identified as a microbial trigger in the pathogenesis of AD. Despite evidence pointing to C. pneumoniae as a pathogenic agent, the mechanisms by which C. pneumoniae contributes to amyloid formation have not been fully elucidated. Therefore, the present study aimed to determine the role of C. pneumoniae in promoting Aβ 1-42 amyloid processing in neuronal cells.

**Methods:** Neuronal cell lines were infected with C. pneumoniae in a Sorvall Legend RT at 750X g for 30 minutes at 20°C. An additional 10,000 cells were grown in media alone as a control and treated with sucrose as described above. Neuronal cells were collected at 24 hrs post-infection and treated with 1% Triton X-100 to lyse the cell membrane. Neuronal cells were then fixed with 4% paraformaldehyde for 20 minutes and then permeabilized with 0.1% Triton X-100 for 15 minutes. Neuronal cell lysates were then subjected to Western blot analysis and quantitated by ELISA.

**Results:** Neuronal cell lines infected with C. pneumoniae showed increased Aβ 1-42 amyloid deposition compared to uninfected neuronal cells. The neuronal cell lines infected with C. pneumoniae showed increased Aβ 1-42 amyloid over the uninfected cells from 24 to 72 hrs post-infection. Western blot analysis confirmed an increase in higher molecular weight bands, and the Western blot analysis showed a 10-fold increase in Aβ 1-42 amyloid deposition in neuronal cells infected with C. pneumoniae compared to uninfected cells. The fluorescence intensity of 1-42 amyloid polyclonal primary antibody (6E10) was labeled with horseradish peroxidase-conjugated goat anti-rabbit secondary antibody for 1 hour at room temperature. The slides were washed in distilled water and cover slipped with anti-fading aqueous mounting media (Biomeda cat# M01). The slides were then visualized under a confocal microscope.

**Conclusions:** These data suggest that infection of neuronal cells with C. pneumoniae Chlamydia pneumoniae should be considered as a potential pathogenic trigger for AD. The present study demonstrates that C. pneumoniae infection of neuronal cells was associated with increased cytotoxicity and neuronal death. Aβ 1-42 amyloid is highest at 24 hour post-infection and the neuronal cell lines infected with C. pneumoniae showed increased Aβ 1-42 amyloid over the uninfected cells from 24 to 72 hrs post-infection. Western blot analysis confirmed an increase in higher molecular weight bands and the Western blot analysis showed a 10-fold increase in Aβ 1-42 amyloid deposition in neuronal cells infected with C. pneumoniae compared to uninfected cells. These data suggest that infection of neuronal cells with C. pneumoniae enhances the processing of Aβ 1-42 amyloid and promotes Aβ 1-42 amyloid production.

**References**