Alzheimer’s Disease (AD) is characterized by amyloid deposition and inflammation. Chlamydia pneumoniae (Cpn) intranasal infection of BALB/c mice has been shown to induce AD-like pathology in brain tissue. Preliminary data had previously been localized. Cpn infected mice also displayed greater antigen burden and amyloid burden relative to titer negative mice. Glial cell activation was readily apparent in the brain where chlamydia and Infection of Mice and Tissue Preparation:

10^5 IFU’s of C Small Weiss strain were used to intranasally infect non-transgenic BALB/c mice with C. pneumoniae (Cpn). Infected mice were sacrificed at 1, 3, and 5 weeks post infection. Tissue was collected for immunohistochemistry and histological staining. Paraformaldehyde-fixed tissue was cut into 20 micron sections and then dehydrated and infiltrated with wax. Sections were cut and mounted onto glass slides for conjugate labeling and immunolocalization of antigens. Congo Red staining was performed to label amyloid deposits. Immunohistochemistry was conducted using primary antibodies 10C-CR2104M1, 10C2104M3, Anti-C. pneumoniae C. trachomatis specific antibody. E and F represent titer positive control tissue which were Cpn-infected. A and C are MoPn stock strain (A and C) or CSW (MoPn in vivo passaged) (B and D) immunolabeled with Cpn-specific antibody (10C-CR2104M1, 10C2104M3) , two sets were labeled with beta-Amyloid Labeling

MoPn stock strain Day 60 p.i. 44.58 11.12 91.58 1.23 38.51 6.16 16. Little CS, Hammond CJ, MacIntyre A, Balin BJ, Appelt DM. Chlamydia pneumoniae induces alzheimer-like amyloid plaques and inflammation in immunoglobulin A deficient mice.

The work presented here continues previous studies involving the BALB/c mouse model of AD-like pathology. We have shown that Chlamydia pneumoniae infection induces AD-like pathology and inflammation in the brain, which may contribute to the development of Alzheimer's disease. Our findings suggest a potential role for Chlamydia pneumoniae in the pathogenesis of Alzheimer's disease, and further investigations are needed to elucidate the mechanisms involved.

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Bibliography