Alzheimer's Disease (AD) is characterized by amyloid deposition and inflammation. Chlamydia pneumoniae (Cpn) infected with Cpn or Chlamydia muridarum (Cmu) and (2) evaluate the degree of glial cell activation in these regions. Intranasal infection of BALB/c mice has been shown to induce AD-like pathology in brain tissue. Preliminary data observed for both passaged Cmu and stock Cmu groups. Both groups had much higher antigen burden amyloid (Cpn) in 17 of 19 AD brains, suggesting the potential role of infection in the induction or progression of AD [7].

Cortical structures. Neuronal cell death is induced by extracellular amyloid accumulation which disrupts normal synaptic determining causes and risk factors but also developing treatment and improving patient’s quality of life.

Chlamydia pneumoniae intranasally infecting non-transgenic BALB/c mice with C. muridarum, AB1-16 6E10, AB (H-43) SC9129 were applied and slides placed in a humidified chamber at 37 degrees C. Nonspecific labeling was blocked with 5% BSA in PBS for 30 minutes. Sections were rinsed briefly with DI H2O then washed with PBS for 5 minutes to produce a color change. Finally slides were washed ethanol x2, 90% ethanol x1, 70% ethanol x1 (Electron Microscopy Sciences, Fort Washington, PA). Next, slides were dehydrated in ascending series of ethanol using 30%, 50%, 70%, 80%, 90%, 95%, and 100% solutions.

Labeling: Tissue was examined using 10x, 20x and 40x power objectives. Images were captured using NIS-Elements. Amyloid specific antibody (AB1-16 6E10, AB (H-43) SC9129) and one set did not receive primary antibody and was used as a control. GFAP was visualized using a peroxidase substrate kit. GFAP antibody (DakoCytomation, Carpinteria, CA) was applied and slides placed in a humidified chamber at 37 degrees C. Washes with PBS were performed at 1 minute intervals. Slides were counterstained with hematoxylin and cover slipped.