Comparison of Chlamydia antigen and AD-like pathology in the brains of BALB/c mice following intranasal infection with Chlamydia muridarum or Chlamydia pneumoniae

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Abstract

Previous research utilizing BALB/c mice inoculated with Chlamydia pneumoniae Cpn) demonstrated AD-like alterations. The purpose of this research was to investigate the pathology induced by Chlamydia muridarum in BALB/c mice. BALB/c mice were inoculated intranasally with C. pneumoniae or C. muridarum (10⁵ CFU/mouse). Brains were harvested and immunostained with 6E10 and/or K15 antibodies. Results indicate that C. pneumoniae induces AD-like pathology in BALB/c mice, while C. muridarum does not. The location and degree of AD-like pathology induced by C. pneumoniae was significantly greater than that induced by C. muridarum.

Introduction

Alzheimer’s disease (AD) is a progressive neurodegenerative disorder characterized by a loss of neurons in the brain, leading to cognitive decline and memory loss. The disease is characterized by the deposition of amyloid plaques and neurofibrillary tangles in brain tissue. These two pathological hallmarks are believed to contribute to the cognitive decline observed in AD patients. Early AD pathology involves the medial temporal lobe, which includes the hippocampus and the amygdala. Decline in cognitive function continues as AD progresses, with the accumulation of amyloid plaques and neurofibrillary tangles in brain tissue.

Methods

Two different lines of mouse adopted Chlamydia pneumoniae (Cpn) antigen and Amyloid Deposition (Dep) induced following infection with Cpn. In contrast with transgenic mice, the use of BALB/c mice more accurately models the predominant sites of infection such as the spleen, aorta, and abdominal lymph nodes. Thus, the ability of Cpn to induce AD-like pathology was investigated using BALB/c mice. Cpn-infected mice were sacrificed at 2, 4, 6, and 8 months post-infection. Brains were harvested and fixed, then sectioned and stained with antibodies specific to amyloid plaques and neurofibrillary tangles. Immunohistochemical analysis was performed using ImageJ software to quantify the degree and location of AD-like pathology.

Results

Chlamydia Labeling

Beta Amyloid Labeling

Conclusions

1. Substantial amyloid and Chlamydia labeling at the 2 month time point following intranasal infection with C. pneumoniae suggests that the Chlamydia infection induces amyloid pathology.

2. Amyloid deposition at the 2 month time point is more intracytoplasmic with Chlamydia muridarum demonstrated that it induced with the bacteria induced AD-like pathology.

3. The substantial increase in amyloid deposition and Chlamydia labeling observed following infection with the C. pneumoniae strain suggests that the degree of pathology induced following infection varies based upon the isolate of chlamydia introduced.

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