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Herpes Simplex Virus 1 and Chlamydia (Chlamydia) pneumoniae promote Aβ 1-42 amyloid processing in murine astrocytes linking an infectious process to Alzheimer's disease

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Abstract

Analytical peptides are a pathological feature of Alzheimer's disease (AD) that are formed by the abnormal degradation of fragments of amyloid precursor protein (APP). The formation of these peptides in rodent models results in the formation of Aβ plaques. We evaluated peptides that are known to be associated with sporadic AD and examined their potential role in sporadic AD. The present study examines the pathological changes associated with these two pathogens. Aβ 1-42 amyloid processing in murine astrocytes linking an infectious process to Alzheimer's disease.

Introduction

Analytical peptides are a pathological feature of Alzheimer's disease (AD) that are formed by the abnormal degradation of fragments of amyloid precursor protein (APP). The formation of these peptides in rodent models results in the formation of Aβ plaques. We evaluated peptides that are known to be associated with sporadic AD and examined their potential role in sporadic AD. The present study examines the pathological changes associated with these two pathogens. Aβ 1-42 amyloid processing in murine astrocytes linking an infectious process to Alzheimer's disease.

Material & Methods

Methods

Uninfected Astrocytes

Astrocytes infected for 24hrs with Herpes Simplex Virus 1 or Chlamydia pneumoniae

Infection

Materials

Chlamydia 61C75 FITC Fitzgerald 1:50

Specifically, the study examines the pathological changes associated with these two pathogens. Aβ 1-42 amyloid processing in murine astrocytes linking an infectious process to Alzheimer's disease.

Results

Table: C. pneumoniae-infected astrocytes. The results are consistent with observations of Aβ 1-42 amyloid deposits in AD brains, as well as developed a BALB/c mouse model that demonstrated infection-induced amyloid plaques.

References


3. Balin, B.J., and Appelt, D. M. (2004). Chlamydia pneumoniae induces deposits of amyloid in areas of the brain typically associated with sporadic AD (Balin et al., 2002). The resulting plaques are of interest to the field of Alzheimer's disease research, as they are a potential indicator of the progression of the disease. The plaques are formed by the accumulation of Aβ peptides, which are derived from the amyloid precursor protein (APP). The formation of Aβ plaques is a hallmark feature of Alzheimer's disease and is believed to play a role in the development of the disease.


7. Balin, B. J., Appelt, D. M., and Balin, B. J. (2004). Chlamydia pneumoniae-infected murine astrocytes demonstrate deposits of amyloid plaques. The results are consistent with observations of Aβ 1-42 amyloid deposits in AD brains, as well as developed a BALB/c mouse model that demonstrated infection-induced amyloid plaques.