Infection of neuronal cells by Chlamydia pneumoniae and Herpes simplex virus type 1 alters expression of genes associated with Alzheimer’s disease

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Infection of neuronal cells by Chlamydia pneumoniae and Herpes simplex virus type 1 alters expression of genes associated with Alzheimer’s disease

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Abstract

Several studies have suggested an infectious etiology for Alzheimer’s disease (AD). We have been investigating a potential role for both Chlamydia pneumoniae and Herpes simplex virus type 1 (HSV-1) in the initiation of sporadic late-onset AD. Our current study focuses on investigation of gene expression using Alzheimer-specific Real-Time PCR microarrays on RNA derived from SKN-MC human neuronal monolayer cell infected with C. pneumoniae and/or HSV-1. There are distinct differences in the patterns of gene regulation seen after infection with HSV-1 alone compared to HSV-1 and C. pneumoniae coinfection. Our data suggest that C. pneumoniae and HSV-1 produce a distinct cellular phenotype as compared to HSV-1 alone. Our data also indicate that the interaction of C. pneumoniae and HSV-1 produces a unique cellular phenotype, as compared to HSV-1 alone. Our data also indicate that the interaction of C. pneumoniae and HSV-1 produces a unique cellular phenotype, as compared to HSV-1 alone.

Introduction

Alzheimer’s disease (AD) is the leading cause of dementia, and while a genetic predisposition can account for a small portion of the cases, the majority of cases are a result of a multifactorial etiology that may include infectious agents. Infection within the central nervous system (CNS) by herpes simplex virus type 1 (HSV-1) and Chlamydia pneumoniae has been suggested to play a role in sporadic AD (Hubal et al., 2004). In the early to middle stages of the disease, HSV-1 and/or HSV-1 viral proteins have been expressed in association with AD. This would provide additional evidence that these pathogens might contribute to or cause progression of the pathology of AD (Balint et al., 2009). Over time, the expression of HSV-1 in Alzheimer’s brain tissue appears to decrease, suggesting that HSV-1 may not be a major contributor to the progression of AD (Balint et al., 2009). Despite the decrease, HSV-1 expression remains detectable in Alzheimer’s brain tissue, and infection with HSV-1 can induce changes in gene expression associated with AD (Balint et al., 2009). Studies have also implicated HSV-1 as a potential pathogen contributing to AD pathology (Hubal et al., 2004). HSV-1 activates and promotes latent herpes simplex virus infection, and for the potential to contribute to or cause progression of AD. HSV-1 infection can result in changes in gene expression associated with AD. It has been shown that HSV-1 can participate in the regulation of viral protein expression, which may lead to the expression of other Aβ-amyloid plaques in the brain (Yamaguchi et al., 2003, Yamaguchi et al., 2003). Furthermore, Herpes simplex virus (HSV) is a known risk factor in the etiology of Alzheimer’s disease (AD). However, the role of HSV-1 infection in the etiology of AD is not well understood.

Although the changes were not dramatic, infection of SKN-MC cells with C. pneumoniae resulted in the up-regulation of numerous genes, especially genes involved in蝙蝠科 and select cell signaling genes. Our data suggest: HSV-1 appears to inhibit C. pneumoniae growth in SKN-MC cells. The decrease in % of cells with multiple C. pneumoniae bacteria may affect the inhibition of C. pneumoniae from spreading within the tissue. The down-regulation of numerous genes in the neuronal disease model by HSV-1 may result in a general ability to repress the expression of numerous other genes. Gene regulation in co-infected monolayers is similar to that seen with HSV-1 alone, as opposed to being interdependent between the two pathogens and HSV-1 and C. pneumoniae and the demonstration that C. pneumoniae and HSV-1 interact to cause the disease. C. pneumoniae infection in SKN-MC cells results in the up-regulation of numerous genes, especially genes involved in oxidative stress. C. pneumoniae can directly contribute to plaque formation, as demonstrated by pathogenic factors of Alzheimer’s disease by balancing expression of genes involved in the processing of β-amyloid. The next study is to assess for a co-infected HSV-1+C. pneumoniae infection in SKN-MC cells, to assess the impact of the two pathogens in the CNS of an individual with Alzheimer’s disease. C. pneumoniae infection in SKN-MC cells results in the up-regulation of numerous genes, especially genes involved in oxidative stress. C. pneumoniae can directly contribute to plaque formation, as demonstrated by pathogenic factors of Alzheimer’s disease by balancing expression of genes involved in the processing of β-amyloid. The next study is to assess for a co-infected HSV-1+C. pneumoniae infection in SKN-MC cells, to assess the impact of the two pathogens in the CNS of an individual with Alzheimer’s disease. 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