Equine herpesvirus type 1 infection induce tau protein phosphorylation and accumulation of hyperphosphorylated tau in cultured neurons

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Equine herpesvirus type 1 (EHV-1) is a main cause of respiratory disease, abortion and myeloencephalopathy in horses. Similarly to other alphaherpesviruses EHV-1 is neurotropic and causes latent infection in the neurons of natural host. Despite the fact that many studies have been devoted to the pathogenesis of various clinical forms of EHV-1 infection, mechanisms of the neuronal damage are not fully understood. In the present work, we examined one of the aspects of these disorders- tau-mediated neurodegeneration as a consequence of its hyperphosphorylation and sequestration in aggregates in infected primary murine neurons. Previous reports mainly focus on those HHV-1 infections which proved that tau protein (a microtubule-associated protein) in important in the neurodegenerative process. It is expressed in central nervous system neurons, where it plays a powerful role in regulating the dynamics of microtubule polymerization. Furthermore, it takes part in regulating axonal diameter, in axonal transport and in neurogenesis. When infected with HHV-1, tau protein may undergo modification, mainly via phosphorylation. As a result, phosphorylated tau protein isoforms aggregate and form neurofibrillary tangles, typical of neurodegenerative diseases called tauopathies. We describe, for the first time, that EHV-1 is capable of intensely modifying in the phosphorylation state of tau and that infection with EHV-1 leads to the accumulation of phosphorylated tau in the cytoplasm of neurons, especially during late hours after infection. This accumulation is dependent on the type of tau phosphorylation and on the time of infection.

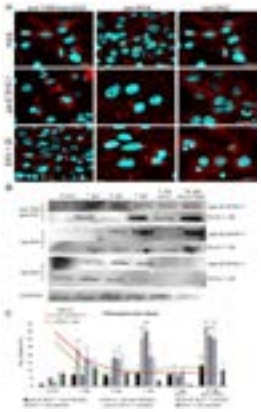

Figure 1: Immunofluorescence and Western blot analysis (A, B) to determine whether EHV-1 infection modifies the phosphorylation state of tau in cultured neurons. Phosphorylated tau levels were quantified by densitometric analysis (C).

Recent Publications

1. Chodkowski M, Serafińska I, Brzezicka J, Golke A, Słońska A, Krzyżowska M, Orłowski P, Bąska P, Bańbura M W and Cymerys J (2018) Human herpesvirus type 1 and type 2 disrupt mitochondrial dynamics in human keratinocytes. Archives of Virology DOI: 10.1007/s00705-018-3890-y.
2. Cymerys J, Słońska A, Tucholska A, Golke A, Chmielewska A and Bańbura M W (2018) Influence of long-term equine herpesvirus type 1 (EHV-1) infection on primary murine neurons - the possible effects of the multiple passages of EHV-1 on its neuro virulence. Folia Microbiologica DOI: 10.1007/s12223-017-0528-5.
3. Cymerys J, Słońska A, Skwarska J and Bańbura M (2016) Function of myosin during entry and egress of equid herpesvirus type 1 in primary murine neurons. Acta Virological 60(4):410-416.
4. Cymerys J, Słońska A, Chodkowski M, Przybylski M and Bańbura M (2016) Primary murine neurons as *in vitro* model for studying neuro infections caused by human adenoviruses. Acta Virological 60(4):417-422.

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Biography

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