Infectious entry of equine herpesvirus-1 into primary murine astrocytes

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Equine herpesvirus type 1 (EHV-1) is a member of the subfamily Alphaherpesvirinae of the family Herpesviridae. In its target host, it induces mild respiratory diseases, abortion, neonatal foal death, and neuropathogenic disorders. EHV-1 has a broad host range in vitro, allowing for study of the mechanisms of productive viral infection, including endocytic pathways or intracellular transport in various cell cultures. The productive infection of astrocytes has been described for HHV-1, HHV-5 and HHV-6; however, there were no data about the ability of EHV-1 to infect the astrocytes. Recently, we reported for the first time that primary murine astrocytes were permissive to EHV-1 infection. Similarly to HHV-1, EHV-1 productively infected astrocytes and displayed cytopathic effect that resulted in the death of a portion of cell population. In the current study we investigated the mechanisms by which EHV-1 enters primary murine astrocytes. Using drugs that inhibit clathrin-dependent (chlorpromazine) or caveola-dependent endocytosis (nystatin), we showed that EHV-1 entry into murine astrocytes require clathrin, but not caveolae. The treatment of the cells with nystatin did not affect the replication of EHV-1. However, the use of chlorpromazine caused a significant decrease in the level of replication of EHV-1 detected by real-time PCR. In conclusion, EHV-1 efficiently entered and replicated in primary murine astrocytes. According to our results, we can assume that the principal pathway of EHV-1 entry into astrocytes appears to be caveolin-dependent and clathrin-independent.

Figure 1: Comparison of viral DNA (CCID50) in astrocytes infected with EHV-1 (Ctrl) and nystatin or chlorpromazine-treated infected primary murine astrocytes (**p<0.01).

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Biography

A Slonska Zielonka, PhD is currently working as Post-doctoral Researcher at Warsaw University of Life Sciences in the Faculty of Veterinary Medicine, Department of Physiological Sciences. She is interested in the field of mechanisms of herpesviral infections in primary murine astrocytes.

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