

## **The effects of the deletion of VHS and EP0 gene on global gene expression of pseudorabies virus**

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Pseudorabies virus (PRV) is a useful model organism for the study of molecular pathogenesis of herpesviruses. This virus has 70 protein coding genes, which belong in three major temporal classes: the immediate-early (IE), early (E) and late (L) classes (the fourth class is the early/late (E/L)). We analyzed the effects of the deletions of virion host shut-off (VHS) and early protein 0 (EP0) genes on the expression kinetics of PRV genes by real-time reverse transcription-PCR. Our data demonstrate that both genes selectively affect the E gene expression of the virus. Our results show that the VHS protein is a coordinator of global gene expression in PRV. This study revealed that in the early stage of viral infection tegument VHS proteins affect the amount of viral transcripts without bias toward any kinetic class of viral transcripts. Later, de novo VHS exerts a differential negative effect on the level of E transcripts. We also found that the effect on the level of L transcripts is slight, and there moderate lowering effect on the level of E/L transcripts. Our data suggest that a major function of the VHS protein is to assist in the switch from the early to late period of infection by selective inhibition of the E transcripts. EP0 exert a selective negative effect on the E transcripts in the early infection period and a general, alternating effect on the amounts of transcripts of each kinetic class of PRV genes in the late phase of infection.

### **Biography**

Dóra Tombác is MSc in Biology (Faculty of Sciences, University of Szeged - 2006) and PhD in Medical Sciences (Faculty of Medicine, University of Szeged - 2010). She works in the Department of Medical Biology at the Faculty of Medicine at University of Szeged in the Boldogkői's group. Their primary field of interest is analysis of herpesvirus gene expression and utilization of herpesviruses as tools in various fields of biology including neurobiology and cardiology.

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