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Opinion Article

The Role of Unfolded Protein Response in Pseudorabies Virus Infection

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ABOUT THE STUDY

The intricate interplay between viruses and host cells has been a subject of intense research, aiming to decipher the molecular mechanisms underlying viral pathogenesis. Among these mechanisms, the Unfolded Protein Response (UPR) has emerged as a pivotal cellular process that safeguards cellular homeostasis and functions as an adaptive stress response. Pseudorabies Virus (PRV), a neurotropic herpes virus affecting a wide range of mammalian species, has been shown to exploit the host cell machinery for its replication and propagation. This study delves into the recent findings elucidating the induction of UPR during PRV infection, shedding light on the intricate crosstalk between viral manipulation and cellular stress response.

Viral infections and cellular stress responses

Viral infections impose a significant burden on host cells, perturbing cellular processes and demanding a comprehensive cellular response. The UPR, a conserved signaling pathway primarily residing in the Endoplasmic Reticulum (ER), is activated in response to ER stress caused by accumulation of unfolded or misfolded proteins. The UPR encompasses three main transducers: Inositol-Requiring Enzyme 1 (IRE1), Protein Kinase RNA-Like ER Kinase (PERK), and Activating Transcription Factor 6 (ATF6), which collectively orchestrate a series of events aimed at restoring ER homeostasis or initiating cell death if stress becomes overwhelming.

PRV and cellular hijacking

PRV, also known as Aujeszky's disease virus, infects the nervous system and causes severe economic losses in the swine industry. Its intricate ability to manipulate the host cell's machinery contributes to viral survival and dissemination. Recent studies have shown that PRV exploits the UPR to its advantage, modulating the ER stress response to create a favorable microenvironment for its own replication. This interaction underscores the remarkable evolutionary adaptation that viruses like PRV have undergone to ensure their survival.

UPR induction during PRV infection

A growing body of evidence supports the notion that PRV infection triggers ER stress, culminating in the activation of UPR signaling pathways. Upon infection, PRV induces the accumulation of viral glycoproteins in the ER, which may lead to protein misfolding and ER stress. This triggers the activation of IRE1, PERK, and ATF6, initiating the UPR cascade. Activation of IRE1 leads to the unconventional splicing of X-Box Binding Protein 1 (XBP1) mRNA, generating a potent transcription factor that up regulates UPR-related genes. Concurrently, PERK activation results in phosphorylation of Eukaryotic Translation Initiation Factor 2α (eIF2α), attenuating global protein synthesis while selectively promoting the translation of specific stress-response genes. Additionally, ATF6 translocates to the nucleus, where it regulates the expression of chaperone proteins, contributing to protein folding and ER-Associated Degradation (ERAD).

Consequences of UPR manipulation

The interplay between PRV and the UPR yields a complex network of molecular events that influence viral replication and cellular fate. The induction of UPR components appears to be a double-edged sword, as it creates an environment conducive to viral replication while also potentially triggering antiviral immune responses. The UPR-mediated up regulation of chaperones and ERAD components assists in folding viral proteins, supporting efficient viral assembly and maturation. Conversely, the UPR may provoke immune recognition through the activation of Pattern Recognition Receptors (PRRs), leading to the production of pro-inflammatory cytokines and interferon's. This intricate balance highlights the dynamic relationship between viral manipulation and cellular stress response.

Therapeutic implications and future directions

The revelation of UPR induction during PRV infection opens doors to novel therapeutic avenues. Targeting the UPR components, such as IRE1 or PERK, could potentially disrupt

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the virus-host interplay and impede viral replication. Moreover, understanding the precise molecular mechanisms governing the UPR-PRV interaction may unveil broader insights into the strategies employed by other viruses to exploit cellular stress responses.

CONCLUSION

In conclusion, the induction of the unfolded protein response during Pseudorabies virus infection underscores the remarkable complexity of virus-host interactions. The UPR serves as a crucial mediator of viral replication and propagation, highlighting the virus's adeptness at co-opting host cellular machinery. Unraveling the intricacies of this interplay not only deepens our understanding of viral pathogenesis but also holds promise for the development of innovative antiviral strategies. As research progresses, we anticipate that further discoveries will shed light on the broader implications of UPR manipulation by viruses and its potential exploitation for therapeutic intervention.