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## HSV-1 herpes virus targets ERAD machinery to assure its replication

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It has been described that during viral infection the protein synthesis is highly increased at the endoplasmic reticulum (ER), condition that in some cases trigger ER stress responses by the accumulation of misfolded proteins. Herpes simplex virus type-1 (HSV-1) is a neurotropic virus that causes a latent persistent infection in humans. It has been postulated that during productive infection herpes virus use the ERAD pathway to avoid the immune response; however, the contribution in the viral replication efficiency is unknown. The aim of this study was to investigate the role of the ERAD enhancer EDEM1, an endoplasmic reticulum lectin protein that works in the recognition of misfolded glycoproteins during productive HSV-1 infection. Human neuroglioma cells (H4) stably expressing either luciferase shRNA or EDEM1 shRNA were infected with HSV-1 wild type or mutant null to ICP0 (protein with E3 ubiquitin ligase activity) or US3 (a protein kinase that modifies host cellular machinery). After infection, levels of EDEM1 were measured by either mRNA (RT-PCR) or protein levels (immunoblotting). Our results showed that HSV-1 infection decreases the levels of EDEM1 to favor viral replication efficiency. The relevance of this study is the elucidation of the contribution of the ERAD pathway in the productive infection of HSV-1 and the possible involvement of ICP0 and/or US3 in this pathway.

### Biography

Carola Otth has research interest on the study of the possible neurodegenerative effects, neuronal dysfunctions and innate immune activation on neurons and astrocytes infected with the neurotropic virus as HSV-1.

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