

Name of Journal: *World Journal of Virology*

ESPS Manuscript NO: 22098

Manuscript Type: Review

Inflammatory and oxidative stress in rotavirus infection

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Abstract

Rotaviruses are the single leading cause of life-threatening diarrhea affecting children under 5 years of age. Rotavirus entry into the host cell seems to occur by sequential interactions between virion proteins and various cell surface molecules. The entry mechanisms seem to involve the contribution of cellular molecules having binding, chaperoning and oxido-reducing activities. It appears to be that the receptor usage and tropism of rotaviruses is determined by the species, cell line and rotavirus strain. Rotaviruses have evolved functions which can antagonize the host innate immune response, whereas are able to induce endoplasmic reticulum (ER) stress, oxidative stress and inflammatory signaling. A networking between ER stress, inflammation and oxidative stress is suggested, in which release of calcium from the ER increases the generation of mitochondrial reactive oxygen species (ROS) leading to toxic accumulation of ROS within ER and mitochondria. Sustained ER stress potentially stimulates inflammatory response through unfolded protein response (UPR) pathways. However, the detailed characterization of the molecular mechanisms underpinning these rotavirus-induced stressful conditions is still lacking. The signaling events triggered by host recognition of virus-associated molecular patterns offers an

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