PRNA1-209

Increased mitochondrial superoxide dismutase expression and lowered production of reactive oxygen species during rotavirus infection

Gac Malgorzata[1], *Bigda Jacek*[2], *Vahlenkamp Thomas*[3]

[1]Friedrich-Loeffler-Institut, Greifswald-Insel Riems, Germany
[2]Intercollegiate Faculty of Biotechnology UG-MUG, Gdańsk, Poland
[3]Institute of Virology, Leipzig, Germany

An important result of multiple viral infections (HIV, HBV, influenza) is the destruction or depletion of antioxidant defenses in infected cells, and subsequent redox imbalance which has profound effects on viral replication. Rotaviruses infect epithelial cells of the small intestine and are responsible for severe diarrhea in infants and substantial economic losses in animal husbandry worldwide. We investigated the oxidant/antioxidant status in rotavirus-infected human colon adenocarcinoma (Caco-2) cell line and African green monkey kidney MA-104 cells. Our results show that within the initial 48 hours of infection the expression of the mitochondrial superoxide dismutase (MnSOD) is significantly increased, which correlates with a decrease in reactive oxygen species production, and with a lack of cellular glutathione depletion. During this period the mitochondria display a hyperpolarization of the inner membrane, which leads to an increased mitochondrial membrane potential. No increase in apoptosis was detected in the infected cultures. In contrast to many viral infections which cause redox imbalance in infected cells, the described virus-host interactions suggest that rotavirus infection does not lead to an induction of oxidative stress. This points to a strategy, unlike other human viruses, to prolong cell survival for accumulation of rotaviral particles in infected cells before cell destruction and virus release.