

12<sup>th</sup> World Congress on

# VIROLOGY

October 16-17, 2017 Baltimore, USA

## Impairment of NK cell activation and function during Herpes B virus infection

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Herpes B virus, endemic in macaques, is a simplex virus like HSV-1 in its disease manifestation in natural hosts. Zoonotic infection of herpes B virus in humans, however, results in 80% fatality without timely antiviral intervention. Our previous studies revealed that herpes B virus infected human cells do not down-regulate MHC Class I molecules on the surface of infected cells, but rather up-regulate NK cell inhibitory MHC Class Ib molecules, HLA-E and HLA-G. This induction of NK cell inhibitory markers is an indicator of impairment of NK cells. Therefore, we hypothesized that herpes B virus escapes natural killer (NK) cell lysis in zoonotically infected humans by inhibiting NK cell activation. To test this hypothesis, we used a cell culture model system to examine whether herpes B virus infected HFF cells and herpes B virus infected HFFs co-cultured with peripheral blood mononuclear cells (PBMCs) generated the production of NK cell-activating cytokines and chemokines. Analysis of results revealed that herpes B virus infection resulted in down-regulation of NK cell-activating cytokines and chemokines, i.e., IFN- $\alpha$ , IP-10, IL-8, and MCP-1. Next, we measured NK cell activity during herpes B virus infection by quantifying the expression of cytotoxic granules (granzyme and perforin) and cytokine (TNF- $\alpha$  and IFN- $\gamma$ ) expression. Our results demonstrated that NK cells with or without stimulation during herpes B virus infection failed to express cytotoxic granules or cytokines supporting our hypothesis that NK cell activity is impaired during human infection. In addition, we also examined herpes B virus titers in the HFF cells co-cultured with IFN- $\alpha$  stimulated or un-stimulated NK cells. Our results suggested that NK cells were incapable of restricting herpes B virus infection even when stimulated with IFN- $\alpha$ . Therefore, these results suggest that NK cells during B virus infection are rendered inactive.

### Biography

Mugdha Vasireddi has her expertise in studying host-pathogen relationship between humans and their pathogens. Her novel cell culture model represents an *in vivo* system that could be utilized, where an appropriate animal model cannot be used to study host immune responses. She is also interested in developing rapid and accurate diagnostics for emerging and re-emerging viruses such as influenza and Zika virus. Currently, she serves as one of the Clinical Lab Directors for National B Virus Resource Center that tests human and non-human primate samples for herpes B virus infection. She is part of a team that is focused on developing and extending diagnostic services for Zika virus detection.

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