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Abstract

The antiviral activity of Type 1 Interferon (IFN) has been extensively studied and recognized, especially in regards to Hepatitis and HIV; however the IFN antiviral activity has not been specifically analyzed in reoviral infection.

In this study, a mouse model of reoviral encephalitis was used to determine the role of Type 1 Interferon (IFN) in host antiviral activity. Six mice deficient in IFN-α/β receptor (IFNAR) function were inoculated with Type 3 Dearing (T3D) mammalian orthoreovirus intracranially at 15 days of age and showed signs of clinical illness, lethargy, hunched posture, and dull hair coat at day 7 post infection. Mice were humanely euthanized and tissues were harvested for histologic evaluation on days 8 and 9 post infection. Pathologic lesions including meningoencephalitis, hydrocephalus, rhinitis, hepatitis, interstitial pneumonia and marked lymphoid depletion were identified on routine histologic exam. Further investigation utilizing immunohistochemistry (IHC) for Reovirus, CD3 cells, B220 cells, and F4/80 was performed on the affected tissues. Reovirus was detected in the brain, liver and lung. In the brain there was a robust T-cell (CD3) and macrophage (F4/80) response. In the spleen Caspase-3 immunohistochemistry confirmed marked apoptosis in the lymphoid tissue. Pathologic lesions were not present in the gastrointestinal tract, heart, or kidneys.

This study shows that IFNAR deficient mice are susceptible to reovirus infection at post natal day 15 with morbidity and mortality due to reovirus induced meningoencephalitis, hepatitis, and interstitial pneumonia.

Keywords

Type 1 Interferon; mammalian orthoreovirus; antiviral response; IFN-α/β

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