Title:	Murine-β-coronavirus infection differently induces "Interferon-induced protein with tetratricopeptide repeats 2 (Ifit2)" in Neuroglial cells
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Abstract:	The Innate immune system in vertebrates is the first line of defence against infectious pathogens and detrimental environmental stress. The host cells' cellular receptors help recognize the offensive agent's chemical nature and trigger a signalling cascade that leads to transcriptional induction of protective proteins. The principle of cell-intrinsic self-defence coupled mechanism to help neighbors best exemplify by interferons (IFN) system, i.e., the first line of defence against viral infection. The genetic material of virus recognized by the cellular receptors like TLRs and RLRs, induces hundreds of interferons and Interferon Stimulated Genes (ISGs). Among many viral stress-inducible proteins, type I IFN is critical. Type I IFN induces antiviral proteins in uninfected cells, many of which are also directly generated in the infected cells. Interferons further induces various type of ISGs, out of many ISGs, IFITs have prominent protective role in viral inhibition. In humans there are four IFIT genes, and mice have three, out of which Ifit2 {Interferon-induced protein with tetratricopeptide repeats 2 (Isg56)} plays a vital role in inhibiting viral replication and regulating cellular functions, but the mechanism of inhibiting viral replication is unclear. This study reviewed the recent findings on the anti-viral role of Ifit2 in murine β-CoV, Mouse hepatitis Virus (MHV-A59) infection in mice. Further, in order to establish a working model to understand how Ifit2 inhibits the viral replication, the first step of my studies is to understand CNS cell-specific expression of Ifit2, upon RSA59 infection in a reductionist in vitro cell culture system. I further studied the regulation of fractalkine CX3CL1 and its receptor CX3CR1 in vitro. My studies showed that primary astrocytes have highest Ifit2 upregulation upon RSA59 infection and can be used an efficient cell culture model to understand the anti-viral mechanism of Ifit2 in murine β-CoV infection.
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