Biological Sciences
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Vesicular Stomatitis Virus Research
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Production of VSV Expression Plasmids to Identify The Viral Component Responsible For IFN Gene Regulation. D.M. Grant, K. Reigle, N. , A.Varble, M.C. Ferran Derik_Grant@urmc.rochester.edu ; mcfsbi@rit.edu

Induction of the cellular interferon response results in the establishment of an antiviral state, preventing spread of a viral infection. Like many other viruses, vesicular stomatitis virus (VSV) has evolved mechanisms that allow it to evade the interferon response, therefore allowing a successfully infection to occur. Previous results generated in our lab indicate that VSV inhibits the IFN response by blocking activation of NF-κB, a transcription factor that is essential for IFN gene induction. In quiescent cells NF-κB is found in the cytoplasm bound to the inhibitory protein, IκB. Upon viral activation, IκB is phosphorylated and degraded, allowing for the nuclear translocation of NF-κB. Once in the nucleus NF-κB activates the IFN promoter, resulting in induction of the IFN gene. The objective of this project is to study the viral component(s) responsible for IFN gene regulation. We have generated eukaryotic expression vectors via recombinant DNA technology. These vectors encode one of the five VSV proteins fused to the green fluorescent protein (GFP), which will allow us to easily identify cells that express the fusion protein. We have been growing up large scale preparations of transfection grade plasmid DNA, which is currently being transfected into mouse L929 cells. NF-κB activation and IFN mRNA production will be examined in transfected cells. We hope that this approach will allow us to identify which VSV protein is responsible for regulation of these important cellular events.