

FATE OF THE INHIBITORY PROTEIN I κ B IN VESICULAR STOMATITIS VIRUS-INFECTED CELLS. *M. Quinn, W. Hammond, M.C. Ferran*, Department of Biological Sciences, MHQ0582@rit.edu, mcfbsbi@rit.edu*

One of the primary host defenses that is triggered in virally-infected cells is the induction of type I interferons (IFN α and β). The IFN response causes infected and surrounding cells to enter into an antiviral state thereby blocking virus replication. In response, many viruses have evolved mechanisms that block the IFN response. The goal of this project was to study the mechanisms used by vesicular stomatitis virus (VSV) to regulate IFN gene expression. When inactive, NF- κ B, a host transcription factor required for induction of the IFN gene, is found in the cytoplasm bound to its inhibitor, I κ B. Upon viral infection, I κ B is phosphorylated and degraded, allowing nuclear translocation of NF- κ B. We are comparing the fate of I κ B in cells infected with the IFN suppressing wild type VSV, and VSV mutants that are defective in their interferon suppressing abilities. The VSV matrix protein (M) has been shown to suppress IFN gene expression. To investigate the potential role of another viral component(s) in the regulation of IFN gene expression, we will clone the viral genes from wild type VSV and an interferon-inducing mutant virus into eukaryotic expression vectors, and compare their DNA sequences. Progress on these experiments will be discussed.