DEGRADATION OF IκB IN VESICULAR STOMATITIS VIRUS-INFECTED L929 CELLS. ***C. Lubking*****,** *Y. Yegiazarova***,** *M.C. Ferran*, Department of Biological Sciences, cml9646@rit.edu, yyegiazarova@yahoo.com, mcfsbi@rit.edu*

One of the primary and most powerful host defenses that is triggered upon viral infection of mammalian cells is known as the interferon (IFN) response. This interferon response involves production of the cytokines IFN α and β, whose cellular effects collectively result in the formation of an "antiviral state." In this antiviral state, host cells limit macromolecular synthesis thereby inhibiting virus infection and replication. To counter this resistance viruses have evolved mechanisms that block the IFN system. For example Vesicular Stomatitis Virus (VSV) bypasses this antiviral defense by suppressing transcription of the IFN gene. Results from our lab suggest that VSV prevents IFN gene induction by regulation of NF-κB activation. NF-κB is normally found in its inactive cytoplasmic form, where it is bound to its inhibitory protein IκB. Viral infection initiates IκB phosphorylation and degradation, allowing nuclear translocation of NF-κB. Once in the nucleus NF-κB, in concert with several other host transcription factors, induces the IFN gene. The goal of this project was to study the mechanisms used by VSV to regulate NF-κB activation. To begin, we compared the fate of IκB-α in L929 cells infected with either an IFN suppressing wild type VSV or VSV mutants defective in IFN suppression. Preliminary results indicate that VSV is not regulating NF-κB activation at the IκB-α level. Experiments are currently being conducted to determine if VSV is altering another step in the pathway that leads to NF-κB activation.