RESTRICTION OF NF-kB NUCLEAR LOCALIZATION BY VESICULAR STOMATITIS VIRUS ***A.J. Varble****, J.W. Jackson, M.C. Ferran**, Department of Biological Sciences ajv8634@rit.edu*

Interferon (IFN) is a key component of a virally-infected cells' innate immune defense. IFN causes surrounding cells to enter an anti-viral state, thereby preventing the spread of viral infection. The overall focus of our work is to further understand IFN gene regulation by Vesicular Stomatitis Virus (VSV) and to identify the viral component(s) responsible for this regulation. Wild type VSV suppresses the IFN response while the T1026R1 mutant induces large amounts of IFN in infected cells. Using immunofluorescence and an Elisa-based Trans AM assay, we have determined that NF-kB activation, a transcription factor that is essential for IFN gene regulation, is regulated in VSV-infected cells. Regulation of NF-kB activation by wild type, T1026R1 and M-defective recombinant viruses has been confirmed using the TransAM assay. In addition, we have generated expression vectors that express a GFP-VSV fusion protein upon transfection into eukaryotic cells. We are currently using these vectors to determine which of the five viral proteins is responsible for NF-kB activation. This research will provide insights into the specific mechanism and proteins that VSV uses to prevent the production of IFN.