IDENTIFICATION OF THE VIRAL COMPONENT(S) RESPONSIBLE FOR NF-κB ACTIVATION IN VESICULAR STOMATITIS VIRUS INFECTED L929 CELLS. ***C. Armbruster****, L. Golebiewski, W. Hammond, M.C. Ferran*, Department of Biological Sciences, cea5972@rit.edu, warren@warrenhammond.com, ZibbelCoot@aol.com, mcfsbi@rit.edu.*

Upon viral infection, mammalian cells may induce the expression of interferon (IFN), a protein that activates a signal transduction cascade that leads to an antiviral state in the cell. Many viruses have evolved mechanisms by which they block IFN production, thereby allowing a productive infection to occur. The viral matrix (M) protein of vesicular stomatitis virus (VSV) nonspecifically suppresses IFN gene expression by inhibiting global host transcription. Our results indicate that VSV may also specifically regulate induction of the IFN gene by regulating activation of a cellular transcription factor called NF-κB. Once activated, NF-κB translocates to the nucleus and becomes part of an enhanceosome complex responsible for IFN gene induction. The focus of this study was to further characterize NF-κB activation in VSV-infected cells and to determine which viral component(s) is responsible for this activation. L929 cells (mouse fibroblast) were infected with IFN-suppressing wild type VSV or various IFN-inducing VSV strains. NF-κB nuclear localization was monitored by immunofluorescence and confocal microscopy. While NF-κB was not activated in wild type-infected cells, we report a profound activation of NF-κB in cells infected with IFN-inducing mutant viruses at approximately 2-2.5 hours postinfection. Preliminary results indicate that the M protein, as well as another viral component, are involved in the regulation of NF-κB activation. In order to identify this second viral protein, we are comparing the genomic sequences of the wild type (IFN suppressing) and mutant (IFN inducing) strains of VSV.