THE VESICULAR STOMATITIS VIRUS MATRIX PROTEIN ALONE IS NOT ABLE TO SUPPRESS IFN MRNA EXPRESSION. ***A.Totten,***
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Interferon, a cytokine produced by virally-infected calls, is able to inhibit viral replication and infection. Wild type (Wt) Vesicular Stomatitis Virus (VSV) suppresses the Interferon-beta (IFN-β) antiviral response, allowing a successful infection to occur. In comparison, mutant T1026R1 (R1), which contains mutations in the M, G, and L genes, is unable to suppress the IFN response. Using quantitative Real-time PCR analysis, we've shown that little to no IFN mRNA is produced in Wt infected cells while high levels of IFN mRNA was produced in R1-infected cells. Moderate levels of IFN mRNA was produced in cells infected with the M-defective r1026M strain, suggesting that the M protein alone may not be sufficient to regulate IFN mRNA expression. To further investigate the components involved in regulation of IFN mRNA production, cells were coinfected with R1 and r1026M. Compared to R1-infected cells, IFN mRNA production was significantly reduced in coinfected cells, further suggesting that a second viral component is involved. To determine if the M protein alone is able to suppress IFN mRNA production, transfection experiments were carried out. Cells were transfected with an Wt M or R1 M expression plasmid via a lipid-based transfection method or an electroporation-based transfection method. Following transfection, IFN was activated by R1 infection and total RNA was isolated. Preliminary results indicate that the M protein alone is not able to suppress IFN mRNA expression in nucleofector-transfected cells, however it does limit IFN mRNA production in lipofetAMINE transfected cells. Further work is necessary, however our results may indicate that the VSV M protein alone is not sufficient to regulate the IFN pathway.