INVESTIGATION OF MAMMALIAN IFITM1 GENES AS VIRAL RESTRICTION FACTORS OF HSV-1 INFECTION.

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Herpes Simplex Virus type 1 (HSV-1) is one of the most prevalent viral infections in humans worldwide. One strategy HSV-1 employs is to establish latency within its host to evade the immune system. Though there are drugs available to treat herpesvirus infections, resistance mutants are continuing to rise and become a problem, indicating a need for new therapeutics. In response to viral infections, host cells produce viral restriction factors to block the virus at various steps in the replication cycle. One key defense mechanism involves the expression of interferons, which enhance the production of restriction factors. Understanding restriction factors may help to identify vulnerabilities in the viral life cycle that can be used as drug targets. Among the viral restriction factors expressedare the interferon-induced transmembrane (IFITM) protein family, which can inhibit entry of enveloped viruses at the cell membrane. Previous research has shown that IFITM1 is the most potent of the IFITM genes at inhibiting HSV-1, but is however less effective at inhibiting other viruses than IFITM3. It is unknown whether other non-human mammalian IFITM1 orthologs are effective at inhibiting HSV-1. We hypothesize that when the IFITM1 gene from cat, cow, goat, and flying fox is overexpressed in Vero cells it will inhibit HSV-1 infection based on similarity at the amino acid level between these genes. Here we report our progress on this project as we construct and verify overexpression plasmids for these experiments. Our next objective is to conduct overexpression and viral challenge experiments using quantitative PCR to measure infectivity.