A Comparative Genomic Analysis of Two Strains of VSV: Searching for the Viral Component(s) Responsible for Interferon Suppression

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Vesicular Stomatisis Virus (VSV) is a member of the rhabdovirus family that primarily targets cattle, although it can infect a very wide array of hosts. VSV contains a negative sense RNA genome that encodes five major proteins: the nucleocapsid protein (N), the phosphoprotein (P), the matrix protein (M), the glycoprotein (G), and the large protein (L), which acts as a viral polymerase. The relatively simple composition of the virus makes VSV an ideal model system for the study of viral-host interactions. With these five proteins, VSV must perform all of its necessary functions, such as replication and the inhibition of host transcription, translation and cellular defenses. The method via which VSV evades one of the primary cellular viral defenses, the Interferon (IFN) response is being investigated. Interferons are a family of proteins, many of which have antiviral properties or act as transcription factors for viral-inhibiting genes. IFN is also secreted from cells, eliciting a similar response in surrounding cells. We are trying to identify the specific viral proteins(s) responsible for the suppression of IFN in infected mouse L929 cells. In order to accomplish this goal, the genomes of two closely related strains of VSV are being sequenced and compared. The major difference between these strains is that one of them fails to effectively suppress interferon until very late in the infection cycle, while the other acts similar to wild type VSV. It is therefore likely that the viral components necessary for inhibiting host IFN's is mutated in some way in the less effective strain of VSV. The entire genomes have not been sequenced yet, but preliminary data appears promising. A mutation has been identified in amino acid 52 of the M protein, a protein which had previously been tied to interferon suppression. This mutation is located in a region that is suspected to contain at least one nuclear localization signal and mutations have been identified in this general region in other viruses that fail to suppress interferon, including the strain T1026R1 which is also studied in our lab. In order to confirm experimentally which viral components are necessary for IFN suppression, The individual genes of various strains of VSV are being cloned into a GFP containing expression vector so that they can be transfected into L929 cells. IFN mRNA production and activation of several transcription factors that are essential for IFN gene induction will be measured in cells expressing various combinations of the VSV genes.

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