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The Herpes Simplex Virus Type-2 UL24 protein: A potential antagonist of the cellular interferon regulatory pathway
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Known as the causative agent of genital herpes, HSV-2 infection affects half a billion people worldwide. When HSV-2 infects a host cell, the virus uses the host cell nucleic acid and protein synthesis machinery to replicate and assemble new viral particles. HSV-2, as well as the numerous other herpesviruses, have been shown to synthesize numerous immune modulation proteins. Some viral proteins work simultaneously to block interferon synthesis and its signaling effects. Since interferon is a critical player in host innate immunity, this may alter the immune response of the host significantly, for example prevent a proper early, innate immune response to infection. However, the HSV-2 UL24 mutant seems to fail in inhibiting IFN production. The mutant was shown to be significantly attenuated for virulence in multiple animal models and also acted as an effective prophylactic vaccine in mammalian models. The proposed project will attempt to elucidate the mechanism through which the HSV-2 UL-24 protein inhibits the IFN regulatory pathway. Western blot and immunofluorescence microscopy of wild type and mutant infected cells will be used to observe potential differences in the expression of a critical host IFN regulatory protein, IRF3. An intimate understanding of how HSV-2 inhibits IFN production and evades the innate immune response may introduce new considerations in the development of HSV-2 vaccine and antiviral therapies.