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*Developing a Complementation System  
for Human Cytomegalovirus (HCMV)  
Mutants Expressing Mutant Forms of  
the Tegument Protein pUL69.*

Human cytomegalovirus (HCMV), a beta herpes virus, is a leading cause of severe birth defects and morbidity and mortality among immunocompromised individuals. Active research is aimed at understanding the mechanism of HCMV replication and pathogenesis. Viral tegument proteins are important structural components of HCMV and can affect viral replication from the onset of infection. pUL69 is one of 25 viral tegument proteins in HCMV. Using a UL69 deletion mutant virus (HCMV  $\Delta$ UL69), researchers have shown that pUL69 plays an important role in the replication and spread of HCMV. Further, *in vitro* studies have attributed several possible functions to pUL69. These possible functions include arresting host cells in the G1 phase of the cell cycle, shuttling between the cytoplasm and nucleus, and interacting with cellular proteins involved in transcription. Several HCMV mutants that are defective for these functions and/or interactions have been generated and some exhibit a similar replication defect as the  $\Delta$ UL69 virus. To aid in the analysis of these mutants my project is to generate a complementation system that will allow for the efficient replication of the UL69 mutant viruses. I will use two distinct techniques. First I will generate a retroviral (MLV) vector and a lentiviral (HIV-1) vector, both which will express UL69. These vectors will be used to generate UL69 expressing, replication-deficient retroviruses that will provide UL69 *in trans* in the target cell. This should complement the  $\Delta$ UL69 replication defect seen in the mutant HCMVs. Second, I will generate a stable cell line that expresses UL69. The data here shows we are able to force expression of UL69 in target cells. Now each technique will be screened for its ability to complement the  $\Delta$ UL69 replication defect.

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