Supporting Information

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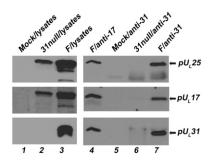


Fig. S1. Coimmunoprecipitation of pU_L17 , pU_L25 , and pU_L31 . CV1 cells were mock infected or were infected with HSV-1(F) or U_L31 null virus. At 18 h after infection, cells were lysed. *Left:* Portions of the lysates were clarified, denatured in SDS, electrophoretically separated, transferred to nitrocellulose, and probed with antibodies to pU_L25 , pU_L17 or pU_L31 . *Right:* Soluble lysates were reacted with pU_L31 -specific antibody, immune complexes were purified, denatured in SDS, and subjected to immunoblotting with anti- pU_L31 , and anti- pU_L17 antibodies.

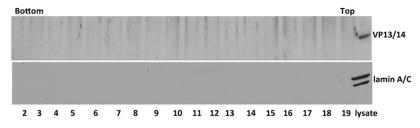


Fig. 52. Immunoblot of capsids separated in sucrose gradients and probed with antibodies to tegument proteins VP13/14 or lamin A/C. Capsids in HSV-1(F)infected lysates were pelleted through a sucrose cushion, resuspended, and then separated by rate zonal centrifugation on two different continuous sucrose gradients. Protein in 0.5-mL TCA precipitated fractions collected from two different gradients were denatured, electrophoretically separated, and transferred to two different nitrocellulose sheets. One sheet was probed with antiserum to the major tegument protein VP13/14 (*Upper*), and the other was probed with monoclonal anti-lamin A/C, a nuclear lamina component (*Lower*). The lysate lanes contain total infected cell lysates solubilized in SDS.