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THE ROLE OF DIVERSE VPU PROTEINS IN HUMAN IMMUNODEFICIENCY VIRUS TYPE 1 (HIV-1) VIRAL PARTICLE RELEASE

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A constant battle is waged between host defenses and viral evasion of those defenses in the human immune system. One such interaction is between the retrovirus human immunodeficiency virus (HIV-1) and a cellular factor which tethers viral particles to the surface of infected cells, inhibiting viral particle release. Vpu, a small transmembrane protein encoded by HIV-1 and some simian immunodeficiency viruses (SIV), enhances viral particle release from infected cells, possibly by inhibition of that tethering factor. Here, we investigated the correlation of Vpu function and location of the virus within the host cell. First, we identified the particle release-enhancing-potential of Vpu proteins from different clades, or strains, of HIV-1 and SIV with naturally varying functional efficiency. Next, we examined the localization of these different Vpu protein within cellular membranes. Vpu has been proposed to localize to organelles including the endoplasmic reticulum, Golgi, and some endosomal compartments that are important in shuttling proteins to and from the cell surface. We fluorescently tagged three such endosomal compartments with red markers and our various Vpu proteins with green markers and examined colocalization. Our results suggest a link between early-endosomal localization and enhancement of release. Additionally, all Vpu proteins localized to the recycling endosome regardless of enhancement of release. These combined data suggest that Vpu may be trafficked through the recycling endosome and early endosome and that Vpu localization within these compartments may be required for Vpu-mediated virus particle release.