Retroviral compatibility with diverse glycoproteins has been known and identified through the course of several studies. However, molecular mechanisms of glycoprotein acquisition are poorly defined. Glycoproteins are acquired by the virus as it buds out of the cell at the plasma membrane. Budding of retroviruses involves multiple interactions between viral and cellular proteins and a mature viral particle is the consummation of a regulated and a sequential process. Currently there are no drugs to target the assembly step of retrovirus.

In the series of studies outlined here, we outline a physical factor, Vpu that contributes to glycoprotein exclusion from HIV particles. Using a model Vpu target, Gibbon ape Leukemia Virus (GaLV) Env, we have deduced the characteristics of a protein that is targeted by Vpu through its cytoplasmic tail domain (CTD). This unique observation of Vpu modulating the GaLV Env CTD allowed us to compare the two modes of Vpu mediated protein modulation- CTD mediated and membrane spanning domain (MSD) mediated.

Subsequently, we studied the contribution of MSD hydrophobicity to Env recruitment to viral budding sites. Curiously, although hydrophobicity of MSD did not dictate Env recruitment, the helicity changes as a result of our mutations resulted in observation of the Env fusogenicity.