Measles Virus (MV) is a highly contagious immunosuppressive disease. MV was attributed to 164,000 deaths in 2008 alone. MV is spread when an infected person coughs or sneezes and an uninfected person inhales the moisture droplets. MV then binds with its receptor, the SLAMF1 molecule, and enters human antigen presenting cells (APCs). Many medically important viruses, such as HIV and *Ebolavirus*, are known to induce host cell signaling upon receptor binding. These signaling pathways facilitate entry and allow the virus to replicate efficiently. Very little is known about the signaling induced when MV binds to its receptor during infection. Our hypothesis is that one or more proteins known to be associated with SLAMF1 signaling are necessary for MV entry into APCs. We first tested an inhibitor of the Src family of kinases. Preliminary data suggests that inhibition of Src kinases reduces the ability of MV to form syncitia in VHS cells that express SLAMF1. We are now using inhibitors to target other known SLAMF1 signaling molecules. In addition to Src, we will also target AMPK and ERK signaling molecules. Once we have collected data with MV pseudovirions, we will apply the inhibitors to APCs and then infect wild-type MV. If our hypothesis is correct, the inhibition of these proteins will also inhibit wild-type MV entry in APCs. These data will be useful in understanding the basic properties associated with MV entry in human immune cells. Additionally, our data could lead to the development of future therapeutics targeted against MV.