Measles is a highly contagious acute respiratory disease caused by the Measles virus (MV) and is characterized by a rash and severe immune suppression. Recent MV outbreaks have underscored the importance of continued study on this virus. MV utilizes human proteins, including SLAMF1, to gain entry into human cells. SLAMF1 can recruit and activate other proteins involved in a process called autophagy. Autophagy involves the recycling of cellular host components within vesicles. The vesicles are easily detected by light microscopy. We will use the VHS cell line that expresses SLAMF1 as the only MV receptor. SLAMF1 is known to directly bind to the MV H protein, allowing entry of the virus. We hypothesize that MV elicits autophagy using SLAMF1. To date, we have shown that binding of the MV-H glycoprotein to SLAMF1 can trigger autophagy in VHS cells. MV-H stimulation of autophagy is greatest at between three and five hours post-viral binding to VHS cells. We are now trying to quantify the number of autophagosomes over a five-hour time course. Drugs that inhibit or stimulate autophagy will be used as negative and positive controls, respectively. Combined, these data have provided preliminary evidence that MV is capable of inducing autophagy through binding to its receptor and is helping expand our current understanding of MV pathogenesis. These findings could aid in developing novel anti-viral therapeutics that target MV replication in host cells early on in infection.