Ebola virus (EBOV) is a deadly pathogen that causes severe hemorrhagic disease in humans and non-human primates. EBOV entry into permissive cells is facilitated by viral glycoproteins (GPs). EBOV-GPs are found on the surface of the virus, and allow viral binding to receptor protein(s) on the host-cell surface. This allows viral uptake into the cell and the viral life cycle to continue. To safely study EBOV entry events, a pseudovirion system is used. This system uses vesicular stomatitis virus (VSV), a cattle virus as a surrogate virus. The VSV surrogate virus does not express its own GP and is therefore replication defective. Because both VSV and EBOV exit a host cell by budding from the host cell plasma membrane, expression of the EBOV-GP in a cell that is also infected with the VSV surrogate virus will create a VSV virion that has the EBOV-GP on its surface (VSV-EBOV-GP). Specifically, we will use the chimeric virus to determine if the EBOV-GP stimulates the cellular process of autophagy. Autophagy is a normal cell process used to discard mis-folded proteins and organelles that have sustained damage. We hypothesize that binding of the EBOV-GP to a host cell stimulates autophagy. We will test this hypothesis by utilizing VSV-EBOV-GP pseudovirions and the “Vero” mammalian cell line. If our hypothesis is correct, we should see the development of autophagic vesicles upon VSV-EBOV-GP binding to Vero cells. The triggering of autophagy by the EBOV-GP could represent a new target for future therapeutics.