

Envelope glycoprotein Recombinant Protein | GP recombinant protein

Recombinant Zaire ebolavirus Envelope glycoprotein

CATALOG # AAA115668	HOST E Coli or Yeast or Baculovirus or Mammalian Cell
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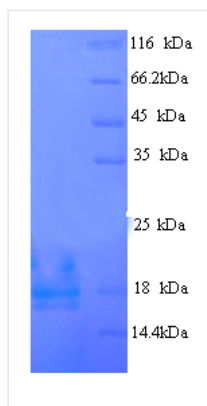
PRODUCT SYNONYMS

Envelope glycoprotein; Recombinant Zaire ebolavirus Envelope glycoprotein; GP1; 2; GP; GP recombinant protein

PRODUCT SPECIFICATIONS

Host	E Coli or Yeast or Baculovirus or Mammalian Cell
Purity/Purification	Greater or equal to 85% purity as determined by SDS-PAGE.
Form/Format	Lyophilized or liquid (Format to be determined during the manufacturing process)
Sequence Positions	502-637
Sequence	EAIVNAQPKC NPNLHYWTTQ DEGAAIGLAW IPYFGPAAEG IYTEGLMHNQ NGLICGLRQL ANETTQALQL FLRATTELRT FSILNRKAID FLLQRWGGTC HILGPDCCIE PHDWTKNITD KIDQIIHDFV DKTLPD
Sequence Length	676
Preparation and Storage	Store at -20 degree C, for extended storage, conserve at -20 degree C or -80 degree C.

IMAGES



SDS-PAGE

ADDITIONAL INFORMATION

Related Product Information for GP recombinant protein	GP1 is responsible for binding to the receptor(s) on target cells. Interacts with CD209/DC-SIGN and CLEC4M/DC-SIGNR which act as cofactors for virus entry into the host cell. Binding to CD209 and CLEC4M, which are respectively found on dendritic cells (DCs), and on endothelial cells of liver sinusoids and lymph node sinuses, facilitate infection of macrophages and endothelial cells. These interactions not only facilitate virus cell entry, but also allow capture of viral particles by DCs and subsequent transmission to susceptible cells without DCs infection (trans infection). Binding to the macrophage specific lectin CLEC10A also ses to enhance virus infectivity. Interaction with FOLR1/folate receptor alpha may be a cofactor for virus entry in some cell types, although results are contradictory. Mbers of the Tyro3 receptor tyrosine kinase family also se to be cell entry factors in filovirus infection. Once attached, the virions are internalized through clathrin-dependent endocytosis and/or macropinocytosis. After internalization of the virus into the endosomes of the host cell, proteolysis of GP1 by two cysteine proteases, CTSB/cathepsin B and CTSL/cathepsin L presumably induces a conformational change of GP2, unmasking its fusion peptide and initiating membranes fusion. GP2 acts as a class I viral fusion protein. Under the current model, the protein has at least 3 conformational states: pre-fusion native state, pre-hairpin intermediate state, and post-fusion hairpin state. During viral and target cell membrane fusion, the coiled coil regions (heptad repeats) assume a trimer-of-hairpins structure, positioning the fusion peptide in close proximity to the C-terminal region of the ectodomain. The formation of this structure appears to drive apposition and subsequent fusion of viral and target cell membranes. Responsible for penetration of the virus into the cell cytoplasm by mediating the fusion of the membrane of the endocytosed virus particle with the endosomal membrane. Low pH in endosomes induces an irreversible conformational change in GP2, releasing the fusion hydrophobic peptide. GP1,2 mediates endothelial cell activation and decreases endothelial barrier function. Mediates activation of primary macrophages. At terminal stages of the viral infection, when its expression is high, GP1,2 down-modulates the expression of various host cell surface molecules that are essential for immune surveillance and cell adhesion. Down-modulates integrins ITGA1, ITGA2, ITGA3, ITGA4, ITGA5, ITGA6, ITGAV and ITGB1. GP1,2 alters the cellular recycling of the dimer alpha-V/beta-3 via a dynamin-dependent pathway. Decrease in the host cell surface expression of various adhesion molecules may lead to cell detachment, contributing to the disruption of blood vessel integrity and hemorrhages developed during Ebola virus infection (cytotoxicity). This cytotoxicity appears late in the infection, only after the massive release of viral particles by infected cells. Down-modulation of host MHC-I, leading to altered recognition by immune cells, may explain the immune suppression and inflammatory dysfunction linked to Ebola infection. Also down-modulates EGFR surface expression. GP2delta is part of the complex GP1,2delta released by host ADAM17 metalloprotease. This secreted complex may play a role in the pathogenesis of the virus by efficiently blocking the neutralizing antibodies that would otherwise neutralize the virus surface glycoproteins GP1,2. Might therefore contribute to the lack of inflammatory reaction seen during infection in spite the of extensive necrosis and massive virus production. GP1,2delta does not se to be involved in activation of primary macrophages.
References	Emergence of subtype Zaire Ebola virus in Gabon.Volchkov V., Volchkova V., Eckel C., Klenk H.-D., Bouloy M., Leguenno B., Feldmann H.Virology 232:139-144(1997)

NCBI AND UNIPROT INFORMATION

NCBI GI #	8479505
UniProt Accession #	P87671
Molecular Weight	17.3 kDa
NCBI Official Full Name	Envelope glycoprotein
UniProt Protein Name	Envelope glycoprotein
UniProt Gene Name	GP
UniProt Synonym Gene Names	GP
UniProt Entry Name	VGP_EBOEC

For Research Use Only. Not for use in diagnostic or therapeutic procedures. Small volumes of product may become entrapped in the seal of the vial during shipment and storage; if necessary, briefly centrifuge the vial on a tabletop centrifuge to dislodge any liquid in the cap.