Product Datasheet

Recombinant Sudan ebolavirus Envelope glycoprotein

Catalog No: #AP73080

Package Size: #AP73080-1 20ug #AP73080-2 100ug #AP73080-3 1mg

Orders: order@signalwayantibody.com
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Description

Product Name	Recombinant Sudan ebolavirus Envelope glycoprotein
Brief Description	Recombinant Protein
Host Species	Yeast
Purification	Greater than 90% as determined by SDS-PAGE.
Immunogen Description	Expression Region:502-637aaSequence Info:Partial
Other Names	GP1,2 ;GP
Accession No.	Q7T9D9
Uniprot	Q7T9D9
GeneID	3160774;
Calculated MW	17.4 kDa
Tag Info	N-terminal 6xHis-tagged
Target Sequence	QTNTKATGKCNPNLHYWTAQEQHNAAGIAWIPYFGPGAEGIYTEGLMHNQNALVCGLRQLANETTQALQLFL RATTELRTYTILNRKAIDFLLRRWGGTCRILGPDCCIEPHDWTKNITDKINQIIHDFIDNPLPN
Formulation	Tris-based buffer50% glycerol
Storage	The shelf life is related to many factors, storage state, buffer ingredients, storage temperature and the stability of the protein itself. Generally, the shelf life of liquid form is 6 months at -20°C,-80°C. The shelf life of lyophilized form is 12 months at -20°C,-80°C.Notes:Repeated freezing and thawing is not recommended. Store working aliquots at 4°C for up to one week.

Background

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. 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 mbranes 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 mbrane 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 mbranes. Responsible for penetration of the virus into the cell cytoplasm by mediating the fusion of the mbrane of the endocytosed virus particle with the endosomal mbrane. 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 horrhages 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

Rapid diagnosis of Ebola hemorrhagic fever by reverse transcription-PCR in an outbreak setting and assessment of patient viral load as a predictor of outcome.Towner J.S., Rollin P.E., Bausch D.G., Sanchez A., Crary S.M., Vincent M., Lee W.F., Spiropoulou C.F., Ksiazek T.G., Lukwiya M., Kaducu F., Downing R., Nichol S.T.J. Virol. 78:4330-4341(2004)Research Topic:Others

Note: This product is for in vitro research use only