Product Data Sheet



Ebola virus Glycoprotein/GP Protein RBD (Q05320, HEK293, Fc)

Cat. No.: HY-P76892

Synonyms: Ebola virus EBOV (subtype Zaire, strain Mayinga 1976) Glycoprotein / GP-RBD Protein (Fc)

Room temperature in continental US; may vary elsewhere.

Species: **HEK293** Source:

Accession: Q05320 (M1-F308)

Gene ID: 911829

PROPERTIES

Molecular Weight: Approximately 57.6 kDa.

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μ m filtered solution of PBS, pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween 80 are added as protectants before lyophilization.
Endotoxin Level	<1 EU/μg, determined by LAL method.
Reconsititution	It is not recommended to reconstitute to a concentration less than 100 μg/mL in ddH ₂ O.
Storage & Stability	Stored at -20°C for 2 years. After reconstitution, it is stable at 4°C for 1 week or -20°C for longer (with carrier protein). It is recommended to freeze aliquots at -20°C or -80°C for extended storage.

DESCRIPTION

Background

Shipping

The trimeric GP1,2 complexes of the Ebola virus Glycoprotein (GP) play a crucial role in viral entry processes, where GP1 serves as the receptor-binding subunit and GP2 acts as the membrane fusion subunit. During later stages of infection, GP down-regulates the expression of various host cell surface molecules, including integrins such as ITGA1, ITGA2, ITGA3, ITGA4, ITGA6, ITGA6, ITGAV, and ITGB1, disrupting cell adhesion and contributing to the detachment of cells, potentially leading to blood vessel integrity disruption and hemorrhages. GP also interacts with host TLR4, stimulating the differentiation and activation of monocytes, resulting in bystander death of T-lymphocytes. Additionally, GP counteracts the antiviral effect of host BST2/tetherin, cooperates with VP40 and host BST2 to activate the canonical NF-kappa-B pathway, and functions as a decoy for anti-GP1,2 antibodies, contributing to viral immune evasion. Moreover, GP interacts with and activates host macrophages and dendritic cells, inducing the up-regulation of cytokine transcription through the activation of host TLR4.

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