

## **Product** Data Sheet

# Ebola virus Glycoprotein/GP1 Protein (Q7T9D9, sf9, His)

Cat. No.: HY-P76884

Synonyms: Ebola virus EBOV (Subtype Sudan, strain Gulu) Glycoprotein / GP1 (mucin domain deleted)

Species: Virus

Sf9 insect cells Source: Accession: Q7T9D9 (M1-D320)

Gene ID: 3160774

Molecular Weight: Approximately 33.8 kDa.

#### **PROPERTIES**

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 $\mu$ m filtered solution of 20 mM Tris, 500 mM NaCl, 10% Glycerol,pH 7.4. Normally 5 % - 8 % trehalose, mannitol and 0.01% Tween80 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 $\mu g/mL$ in ddH <sub>2</sub> 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.
Shipping	Room temperature in continental US; may vary elsewhere.

### **DESCRIPTION**

Background

The trimeric GP1,2 complexes of the Ebola virus Glycoprotein (GP) play a pivotal role in viral entry processes, where GP1 serves as the receptor-binding subunit and GP2 acts as the membrane fusion subunit. In the later stages of infection, GP1 down-regulates the expression of crucial host cell surface molecules involved in immune surveillance and cell adhesion. This includes the modulation of integrins such as ITGA1, ITGA2, ITGA3, ITGA4, ITGA5, ITGA6, ITGAV, and ITGB1, potentially leading to cell detachment and contributing to the disruption of blood vessel integrity, resulting in hemorrhages during infection (cytotoxicity). Additionally, GP1 interacts with host TLR4, stimulating the differentiation and activation of monocytes, leading to bystander death of T-lymphocytes. It further down-regulates the function of host natural killer cells and counteracts the antiviral effect of host BST2/tetherin, which restricts the release of progeny virions from infected cells. Interestingly, GP1 cooperates with VP40 and host BST2 to activate the canonical NF-kappa-B pathway in a manner dependent on neddylation. Furthermore, GP1 functions as a decoy for anti-GP1,2 antibodies, contributing to viral immune evasion, and interacts with host macrophages and dendritic cells, inducing the up-regulation of cytokine transcription, with this effect mediated through the activation of host TLR4.

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