glycoprotein/G Protein, HRSV (258a.a, HEK293, His)

Cat. No.:	HY-P75818
Synonyms:	Human respiratory syncytial virus (RSV) Glycoprotein G Protein
Species:	Virus
Source:	HEK293
Accession:	AII22117 (S64-K321)
Gene ID:	/
Molecular Weight:	Approximately 29.59 kDa

PROPERTIES

Appearance	Lyophilized powder.
Formulation	Lyophilized from a 0.2 μm 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_2O.
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 G protein backbone contains 289 to 299 amino acids (32-33 kDa), depending on the strain, and is palmitoylated. It has no sequence homology with other paramyxovirus attachment proteins, and no hemagglutinating or neuraminidase functions. The G protein has 30-40 O-linked glycans and 4-5 N-linked glycans. The central region of the G protein contains a 13-amino acid highly conserved domain, partially overlapping the cysteine noose domain with 4 cysteines linked 1-4 and 2-3, followed by a highly basic heparin-binding domain (HBD). The HBD is the likely attachment site for heparan sulfate (HS) found on the surface of most cells. A peptide from the G protein HBD (amino acids 184-198) binds efficiently to HEp-2 cells and inhibits RSV infection[1]. The G protein is ~32 kDa without post-translational processing, ~95 kDa when fully glycosylated in Hep-2 cells, ~55 kDa cells after glycosylation and cleavage in Vero cells, and ~170 kDa after post-translational processing in primary human bronchial epithelial cells. G plays an important role in the first step of infection, binding to cell surface molecules through GAGs and/or CX3CR1. Second, G modulates host responses to infection which, in turn, affect immunity and disease. Third, the G-CX3CR1 interaction contributes to both binding to cells and modulating the host response to infection[2].

Caution: Product has not been fully validated for medical applications. For research use only.

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