SARS-CoV S Protein RBD (HEK293, Fc)

Cat. No.:	HY-P73393
Synonyms:	Spike glycoprotein; S glycoprotein; Peplomer protein; S
Species:	Virus
Source:	HEK293
Accession:	AAX16192.1 (R306-F527)
Gene ID:	/
Molecular Weight:	Approximately 51.40 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 SARS-CoV Spike glycoprotein (S) has three subunits S1, S2' and S2 through alternative splicing. S1 can attaches the virion to the cell membrane by interacting with host receptor, initiating the infection. S2' acts as a viral fusion peptide which is unmasked following S2 cleavage occurring upon virus endocytosis. S2 mediates fusion of the virion and cellular membranes by acting as a class I viral fusion protein. Under the current model, S protein has at least three 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. S protein orchestrates viral entry by attaching the virion to the cell membrane through interactions with human ACE2 and CLEC4M/DC-SIGNR receptors. It down-regulates host tetherin (BST2) via lysosomal degradation, countering its antiviral activity. Following attachment, internalization into host cell endosomes induces S glycoprotein conformational changes, potentially unmasking the fusion peptide of S2 through cathepsin CTSL proteolysis. S protein also impairs target cell killing and cytokine production[1][2].

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

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