

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike protein		
	SARS-CoV-2 Spike (Val16-Lys1211)(Thr478Lys, Asp614Gly, Pro681His, Thr732Ala)(Arg682Ser, Arg685Ser, Lys986Pro, Val987Pro) Accession # NP_009724390.1	GCN4-IZ	6-His tag
	N-terminus		C-terminus
N-terminal Sequence Analysis	Val16		
Predicted Molecular Mass	138 kDa		

SPECIFICATIONS

SDS-PAGE	150-170 kDa, under reducing conditions.
Activity	Measured by its binding ability in a functional ELISA with Recombinant Human ACE-2 His-tag (Catalog # 933-ZN).
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 500 µg/mL in PBS.
Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 3 months, -20 to -70 °C under sterile conditions after reconstitution.

DATA

<p>Binding Activity</p> <p>Recombinant SARS-CoV-2 B.1.1.519 Spike GCN4-IZ Protein Binding Activity Recombinant SARS-CoV-2 B.1.1.519 Spike His-tag (Catalog # 10883-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.</p>	<p>SDS-PAGE</p> <p>Recombinant SARS-CoV-2 B.1.1.519 Spike (GCN4-IZ) Protein SDS-PAGE 2 µg/lane of Recombinant SARS-CoV-2 B.1.1.519 Spike (GCN4-IZ) His-tag Protein (Catalog # 10883-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 150-170 kDa.</p>
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BACKGROUND

SARS-CoV-2, which causes the global pandemic coronavirus disease 2019 (Covid-19), belongs to a family of viruses known as coronaviruses that also include MERS-CoV and SARS-CoV-1. Coronaviruses are commonly comprised of four structural proteins: Spike protein (S), Envelope protein (E), Membrane protein (M) and Nucleocapsid protein (N) (1). The SARS-CoV-2 S protein is a glycoprotein that mediates membrane fusion and viral entry. The S protein is homotrimeric, with each ~180-kDa monomer consisting of two subunits, S1 and S2 (2). In SARS-CoV-2, as with most coronaviruses, proteolytic cleavage of the S protein into S1 and S2 subunits is required for activation. The S1 subunit is focused on attachment of the protein to the host receptor while the S2 subunit is involved with cell fusion (3-5). The S protein of SARS-CoV-2 shares 75% and 29% amino acid sequence identity with S protein of SARS-CoV-1 and MERS, respectively. The S Protein of the SARS-CoV-2 virus, like the SARS-CoV-1 counterpart, binds a metalloproteinase, Angiotensin-Converting Enzyme 2 (ACE-2), but with much higher affinity and faster binding kinetics through the receptor binding domain (RBD) located in the C-terminal region of S1 subunit (6). It has been demonstrated that the S Protein can invade host cells through the CD147/EMMPRIN receptor and mediate membrane fusion (7, 8). Polyclonal antibodies to the RBD of the SARS-CoV-2 protein have been shown to inhibit interaction with the ACE-2 receptor, confirming RBD as an attractive target for vaccinations or antiviral therapy (9). There is also promising work showing that the RBD may be used to detect presence of neutralizing antibodies present in a patient's bloodstream, consistent with developed immunity after exposure to the SARS-CoV-2 (10). Several emerging SARS-CoV-2 genomes have been identified with mutations compared to the Wuhan-Hu-1 SARS-CoV-2 reference sequence. The B.1.1.519 variant was identified as becoming the dominant variant in Mexico in early 2021 and it contains several mutations of interest that effect viral fitness and transmissibility: T478K, P681H, and T732A (11). The T478K mutation is located in the RBD and has been identified as a potentially crucial mutation as it may alter hACE-2 binding and mutations at position T478 have shown resistance to neutralization by multiple monoclonal antibodies (12, 13). The P618H mutation is found adjacent to the furin cleavage site and is proposed to enhance S protein cleavage and increase viral infectivity (14).

References:

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