

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike protein		
	SARS-CoV-2 Spike (Val16-Lys1211)(Asp614Gly)(Arg682Ser, Arg685Ser, Lys986Pro, Val987Pro) Accession # YP_009724390.1	GCN4-IZ	6-His tag
	N-terminus		C-terminus
N-terminal Sequence Analysis	Val16		
Predicted Molecular Mass	138 kDa		

SPECIFICATIONS

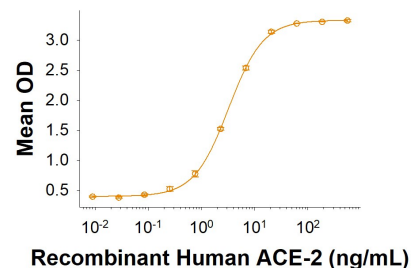
SDS-PAGE	140-170 kDa, under reducing conditions.
Activity	Measured by its binding ability in a functional ELISA with Recombinant Human ACE-2 His-tag (Catalog # 933-ZN).
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>90%, by SDS-PAGE visualized with Silver Staining and quantitative 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 at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	<p>Use a manual defrost freezer and avoid repeated freeze-thaw cycles.</p> <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.

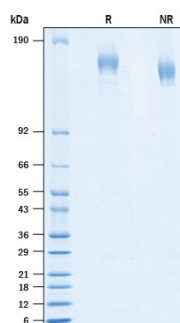
DATA

Binding Activity



Recombinant SARS-CoV-2 D614G Spike (GCN4-IZ) His-tag Protein Binding Activity
Recombinant SARS-CoV-2 Spike (GCN4-IZ) His-tag (Catalog # 10853-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.

SDS-PAGE



Recombinant SARS-CoV-2 D614G Spike (GCN4-IZ) His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant SARS-CoV-2 D614G Spike (GCN4-IZ) His-tag Protein (Catalog # 10853-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 140-170 kDa.

BACKGROUND

SARS-CoV-2, which causes the global pandemic coronavirus disease 2019 (Covid-19), belongs to a family of viruses known as coronaviruses that are commonly comprised of four structural proteins: Spike protein (S), Envelope protein (E), Membrane protein (M), and Nucleocapsid protein (N) (1). SARS-CoV-2 Spike Protein (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 the 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). A SARS-CoV-2 variant carrying the S protein amino acid (aa) change D614G has become the most prevalent form in the global pandemic and has been associated with greater infectivity and higher viral load (6,7). The S protein of SARS-CoV-2 shares 75% and 29% aa 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 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 (8). Based on structural biology studies, the RBD can be oriented either in the up/standing or down/lying state with the up/standing state associated with higher pathogenicity (9). 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 (10). It has been demonstrated that the S Protein can invade host cells through the CD147/EMMPRIN receptor and mediate membrane fusion (11, 12). While the SARS-CoV-2 D614G variant is currently the most prevalent form of the virus, the mechanism of action has not been identified (13).

References:

1. Wu, F. *et al.* (2020) *Nature* **579**:265.
2. Tortorici, M.A. and D. Veasler (2019) *Adv. Virus Res.* **105**:93.
3. Bosch, B.J. *et al.* (2003) *J. Virol.* **77**:8801.
4. Belouzard, S. *et al.* (2009) *Proc. Natl. Acad. Sci.* **106**:5871.
5. Millet, J.K. and G.R. Whittaker (2015) *Virus Res.* **202**:120.
6. Korber, *et al.* (2020) *Cell* **182**:812.
7. Zhang, L. *et al.* (2020) *Nat. Commun.* **11**:6013.
8. Ortega, J.T. *et al.* (2020) *EXCLI J.* **19**:410.
9. Yuan, Y. *et al.* (2017) *Nat. Commun.* **8**:15092.
10. Jiang, S. *et al.* (2020) *Trends Immuno.* **41**:355.
11. Wang, X. *et al.* (2020) *Cell Mol. Immunol.* <https://doi.org/10.1038/s41423-020-0424-9>.
12. Wang, K. *et al.* (2020) *Sig. Transduct Target Ther.* **5**:283.
13. Isabel, *et al.* (2020) *Sci. Rep.* **10**:14031.