

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike protein		
	<p>SARS-CoV-2 BA.2 Spike (Val16-Lys1211) (Thr19Ile, Leu24del, Pro25del, Pro26del, Ala27Ser, Gly142Asp, Val213Gly, Gly339Asp, Ser371Phe, Ser373Pro, Ser375Phe, Thr376Ala, Asp405Asn, Arg408Ser, Lys417Asn, Asn440Lys, Ser477Asn, Thr478Lys, Glu484Ala, Gln493Arg, Gln498Arg, Asn501Tyr, Tyr505His, Asp614Gly, His655Tyr, Asn679Lys, Pro681His, Asn764Lys, Asp796Tyr, Gln954His, Asn969Lys) (Arg682Ser, Arg685Ser, Lys986Pro, Val987Pro) Accession # YP_009724390.1</p>	GCN4-IZ	6-His tag
	N-terminus		C-terminus

N-terminal Sequence Val16

Analysis

Predicted Molecular Mass 138 kDa

SPECIFICATIONS

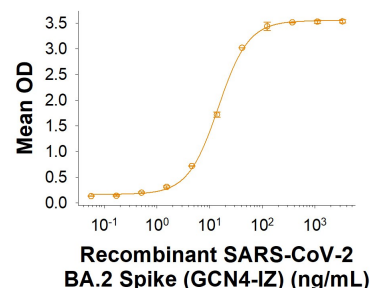
SDS-PAGE	144-172 kDa, under reducing conditions.
Activity	Measured by its binding ability in a functional ELISA with Recombinant Human ACE-2 Fc Chimera (Catalog # 10544-ZN).
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and 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 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.

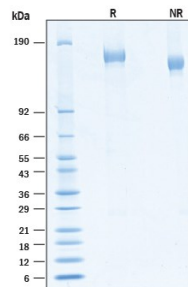
DATA

Binding Activity



Recombinant SARS-CoV-2 BA.2 Spike (GCN4-IZ) His-tag Protein Binding Activity. Recombinant SARS-CoV-2 BA.2 Spike (GCN4-IZ) His-tag Protein (Catalog # 11109-CV) binds Recombinant Human ACE-2 Fc Chimera (Catalog # 10544-ZN) in a functional ELISA.

SDS-PAGE



Recombinant SARS-CoV-2 BA.2 Spike (GCN4-IZ) His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant SARS-CoV-2 BA.2 Spike (GCN4-IZ) His-tag Protein (Catalog # 11109-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 144 - 172 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 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% 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 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 including the Omicron, or B.1.1.529, variant. First identified in November 2021 in South Africa, the Omicron variant quickly became the predominant SARS-CoV-2 variant and is considered a variant of concern (VOC). The Omicron variant contains 32 mutations in the S protein, 3 to 4 times more than in other SARS-CoV-2 variants, that potentially affect viral fitness and transmissibility (11). Of these mutations, 15 are located in the RBD domain and allow the Omicron variant to bind ACE-2 with greater affinity and, potentially, increased transmissibility (11, 12). Several additional mutations throughout the S protein have been shown or are predicted to enhance spike cleavage and could aid transmission (13-15). The study of the Omicron variant's impact on immune escape and reduced neutralization activity to monoclonal antibodies along with an increased risk of reinfection, even among vaccinated individuals, remains ongoing (16). The BA.2 subvariant is predicted to be up to about 35 percent more transmissible than the original Omicron variant..

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. Ortega, J.T. *et al.* (2020) *EXCLI J.* **19**:410.
7. Wang, K. *et al.* (2020) *bioRxiv* <https://www.biorxiv.org/content/10.1101/2020.03.14.988345v1>.
8. Isabel, S. *et al.* (2020) *Sci Rep.* **10**, 14031. <https://doi.org/10.1038/s41598-020-70827-z>.
9. Tai, W. *et al.* (2020) *Cell. Mol. Immunol.* **17**:613.
10. Okba, N. M. A. *et al.* (2020). *Emerg. Infect. Dis.* <https://doi.org/10.3201/eid2607.200841>.
11. Shah, M. and Woo, H.G. (2021) *bioRxiv* <https://doi.org/10.1101/2021.12.04.471200>.
12. Lupala, C.S. *et al.* (2021) *bioRxiv* <https://doi.org/10.1101/2021.12.10.472102>.
13. Zhang, L. *et al.* (2020) *Nat Commun.* **11**:6013.
14. Lasek-Nesselquist, E. *et al.* (2021) *medRxiv* <https://doi.org/10.1101/2021.03.10.21253285>.
15. Scheepers, C. *et al.* (2021) *medRxiv* <https://doi.org/10.1101/2021.08.20.21262342>.
16. Callaway, E. and Ledford, H. (2021) *Nature* **600**:197.