Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag

Catalog Number: 11138-CV

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike S1 Subunit protein Val16-Pro681 (Leu18Phe, Thr20Asn, Pro26Ser, Asp138Tyr, Arg190Ser, Lys417Thr, Glu484Lys, Asn501Tyr, Asp614Gly, His655Tyr) with a C-terminal 6-His tag Accession # YP_009724390.1
N-terminal Sequence Analysis	Val16
Predicted Molecular Mass	75 kDa

SPECIFICATIONS

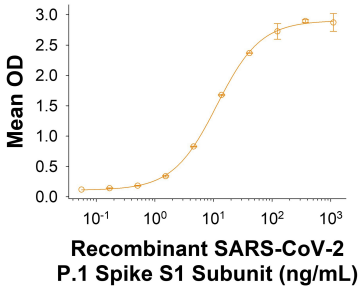
SDS-PAGE	100-120 kDa, under reducing conditions.
Activity	Measured by its binding ability in a functional ELISA with Recombinant Human ACE-2 Fc Chimera (Catalog # 10544-ZN).
Endotoxin Level	<0.10 EU per 1 µg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and 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 200 µg/mL in PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	**Use a manual defrost freezer and avoid repeated freeze-thaw cycles.** • 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.

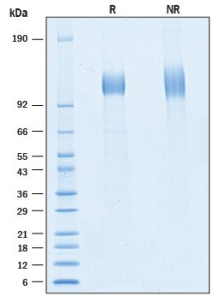
DATA

Binding Activity



Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag Protein Binding Activity. Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag Protein (Catalog # 11138-CV) binds Recombinant Human ACE-2 Fc Chimera (Catalog # 10544-ZN) in a functional ELISA.

SDS-PAGE



Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag Protein (Catalog # 11138-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 100 - 120 kDa.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449

Recombinant SARS-CoV-2 P.1 Spike S1 Subunit His-tag

Catalog Number: 11138-CV

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 commonlycomprised 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 two distinct peptides, 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 metallopeptidase, angiotensin-converting enzyme 2 (ACE-2), has been identified as a functional receptor for SARS-CoV-2 through interaction with a receptor binding domain (RBD) located at the C-terminus of S1 subunit (6, 7). The S1 subunit of SARS-CoV-2 shares 65% amino acid (aa) sequence identity with the S1 subunit of SARS-CoV-1, but only 22% aa sequence identity with the S1 subunit of MERS-CoV. The differences in aa sequence identity is consistent with the finding that SARS and MERS bind different cellular receptors (8). The S Protein of the SARS-CoV-2 virus binds ACE-2 with higher affinity and faster binding kinetics than its SARS-CoV-1 counterpart (9). Before binding to the ACE-2 receptor, structural analysis of the S1 trimer shows that only one of the three RBD domains in the trimeric structure is in the "up" conformation. This is an unstable and transient state that passes between trimeric subunits but is nevertheless an exposed state to be targeted for neutralizing antibody therapy (10). Polyclonal antibodies to the RBD of the SARS-CoV-2 S1 subunit have been shown to inhibit interaction with the ACE-2 receptor, confirming RBD as an attractive target for vaccinations or antiviral therapy (11). 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 virus (12). Lastly, it has been demonstrated the S Protein can invade host cells through the CD147/EMMPRIN receptor and mediate membrane fusion (13, 14). Several emerging SARS-CoV-2 genomes have been identified including the P.1 (Brazilian) variant. The P.1 variant contains numerous mutations of interest in the S1 domain, including 3 mutations in the RBD: K417T, E484K, and N501Y (15). Both the K417T and N501Y mutations have shown increased infectivity and caused reduced neutralization activity to several monoclonal antibodies (16, 17). The E484K mutation is a potentially crucial mutation as it creates a new site for hACE-2 binding and may enhance binding affinity (18). Further, the E484K substitution alone has been shown to confer resistance to several monoclonal antibodies and is responsible for the first confirmed SARS-CoV-2 reinfection (19). Located nearby to the RBD domain, the D614G mutation has been shown to increase viral infectivity (17).

References:

1. Wu, F. *et al.* (2020) Nature **579**:265.
2. Tortorici, M.A. and D. Veesler (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. Li, W. *et al.* (2003) Nature 426:450.
7. Wong, S.K. *et al.* (2004) J. Biol. Chem. **279**:3197.
8. Jiang, S. *et al.* (2020) Trends. Immunol. https://doi.org/10.1016/j.it.2020.03.007.
9. Ortega, J.T. *et al.* (2020) EXCLI J. **19**:410.
10. Wrapp, D. *et al.* (2020) Science **367**:1260.
11. Tai, W. *et al.* (2020) Cell. Mol. Immunol. https://doi.org/10.1016/j.it.2020.03.007.
12. Okba, N.M.A. *et al.* (2020) Emerg. Infect. Dis. https://doi.org/10.3201/eid2607.200841.
13. Wang, X. et al. (2020) https://doi.org/10.1038/s41423-020-0424-9.
14. Wang, K. et al. (2020) bioRxiv https://www.biorxiv.org/content/10.1101/2020.03.14.988345v1.
15. Imai, M. *et al.* (2021) PNAS **118**:e2106535118.
16. Zahradník, J. *et al.* (2021) bioRxiv https://doi.org/10.1101/2021.01.06.425392.
17. Zhang, L. *et al.* (2020) Nat Commun. **11**:6013.
18. Wang, W.B. *et al.* (2021) bioRxiv https://doi.org/10.1101/2021.02.17.431566.
19. Nonaka, C.K.V. *et al.* (2021) Emerg Infect Dis. https://doi.org/10.3201/eid2705.210191.

bio-techne®
Global bio-techne.com info@bio-techne.com techsupport@bio-techne.com TEL +1 612 379 2956
USA TEL 800 343 7475 Canada TEL 855 668 8722 China TEL +86 (21) 52380373
Europe | Middle East | Africa TEL +44 (0)1235 529449