

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived sars-cov-2 Spike S1 Subunit protein
Val16-Pro681, with a C-terminal 6-His tag
Accession # YP_009724390.1

N-terminal Sequence Analysis Val 16

Predicted Molecular Mass 75 kDa

SPECIFICATIONS

SDS-PAGE 105-125 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.

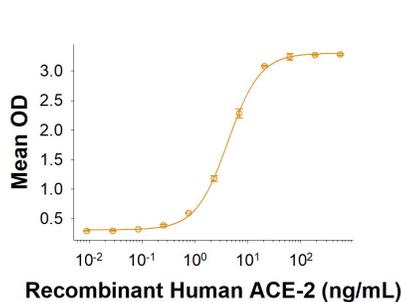
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

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.

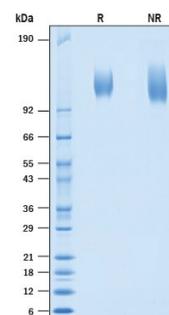
DATA

Binding Activity



Recombinant SARS-CoV-2 Spike S1 Subunit His-tag Protein Binding Activity.
Recombinant SARS-CoV-2 Spike S1 Subunit His-tag (Catalog # 10693-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.

SDS-PAGE



Recombinant SARS-CoV-2 Spike S1 Subunit His-tag Protein SDS-PAGE. 2 µg/lane of Recombinant SARS-CoV-2 Spike S1 Subunit His-tag (Catalog # 10693-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 105-125 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 S1 domain of SARS-CoV-2 S1 share 65% amino acid (aa) sequence identity with the SARS-CoV-1 S1 subunit, but only 22% aa sequence identity with the MERS-CoV S1 subunit. A receptor binding domain (RBD) in the C-terminus of the S1 subunit has been identified and the low aa sequence identity of the S1 subunits is consistent with the finding that SARS and MERS bind different cellular receptors (6). The SARS-CoV-2 virus, like the SARS-CoV-1 counterpart, binds Angiotensin-Converting Enzyme 2 (ACE2), but with much higher affinity and faster binding kinetics (7). Before binding to the ACE2 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 (8). Polyclonal antibodies to the RBD of the SARS-CoV-2 S1 subunit have been shown to inhibit interaction with the ACE2 receptor, confirming the RBD, and the S1 subunit, as an attractive target for vaccinations or antiviral therapy (9). There is also promising work showing that the RBD may be used to detect the presence of neutralizing antibodies present in a patient's bloodstream, consistent with developed immunity after exposure to the SARS-CoV-2 virus (10). Further, it has been demonstrated the S Protein can invade host cells through the CD147/EMMPRIN receptor and mediate membrane fusion (11, 12).

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. Jiang, S. *et al.* (2020) *Trends. Immunol.* <https://doi.org/10.1016/j.it.2020.03.007>.
7. Ortega, J.T. *et al.* (2020) *EXCLI J.* **19**:410.
8. Wrapp, D. *et al.* (2020) *Science* **367**:1260.
9. Tai, W. *et al.* (2020) *Cell. Mol. Immunol.* <https://doi.org/10.1016/j.cmi.2020.03.007>.
10. Okba, N.M.A. *et al.* (2020). *Emerg. Infect. Dis.* <https://doi.org/10.3201/eid2607.200841>.
11. Wang, X. *et al.* (2020) <https://doi.org/10.1038/s41423-020-0424-9>.
12. Wang, K. *et al.* (2020) *bioRxiv* <https://www.biorxiv.org/content/10.1101/2020.03.14.988345v1>.