

## DESCRIPTION

<b>Source</b>	Chinese Hamster Ovary cell line, CHO-derived sars-cov Spike S1 Subunit protein		
	SARS-CoV Spike S1 Subunit (Ser14-Leu666) Accession # YP_009825051.1	IEGRMD	Human IgG <sub>1</sub> (Pro100-Lys330)
	N-terminus		C-terminus
<b>N-terminal Sequence Analysis</b>	Ser14		
<b>Predicted Molecular Mass</b>	99 kDa		

## SPECIFICATIONS

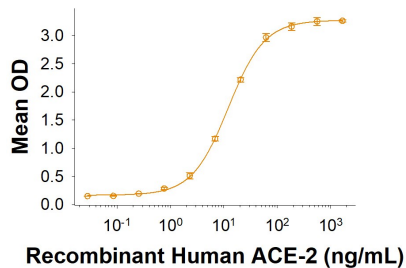
<b>SDS-PAGE</b>	127-141 kDa, under reducing conditions
<b>Activity</b>	Recombinant SARS-CoV Spike S1 Subunit Fc Chimera (Catalog # 10782-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.
<b>Endotoxin Level</b>	<0.10 EU per 1 µg of the protein by the LAL method.
<b>Purity</b>	>95%, by SDS-PAGE visualized with Silver Staining and quantitative densitometry by Coomassie® Blue Staining.
<b>Formulation</b>	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.

## PREPARATION AND STORAGE

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

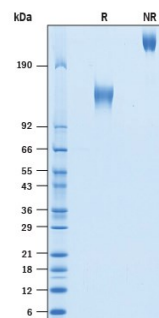
## DATA

### Binding Activity



**Recombinant SARS-CoV Spike S1 Subunit Fc Chimera Protein Binding Activity.**  
Recombinant SARS-CoV Spike S1 Subunit Fc Chimera (Catalog # 10782-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.

### SDS-PAGE



**Recombinant SARS-CoV Spike S1 Subunit Fc Chimera Protein SDS-PAGE.**  
2 µg/lane of Recombinant SARS-CoV Spike S1 Subunit Fc Chimera (Catalog # 10782-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 127-141 kDa and 254-282 kDa, respectively.

## BACKGROUND

SARS-CoV was discovered in association with cases of severe acute respiratory syndrome (SARS) that infected more than 8,000 persons with over 900 fatalities worldwide in 2002-2003 (1). It belongs to a family of viruses known as coronaviruses that also include MERS and SARS-Cov2 that causes the global pandemic coronavirus disease 2019 (Covid-19). Coronavirus is commonly comprised of four structural proteins: Spike protein(S), Envelope protein (E), Membrane protein (M), and Nucleocapsid protein (N) (1). SARS-CoV S Protein is a type-I trimerized membrane glycoprotein that mediates membrane fusion and viral entry. 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 (2-4). A metalloproteinase, angiotensin-converting enzyme 2 (ACE-2), has been identified as a functional receptor for SARS-CoV through interaction with a receptor binding domain (RBD) located at the C-terminus of S1 subunit (5, 6). Based on amino acid (aa) sequence homology, the S1 subunit of SARS-Cov shares 65% and 24% homology with S1 subunit of SARS-CoV2 and MERS, respectively. 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 (7). Antibodies to S protein especially the S1 subunit of SARS-CoV have been shown to inhibit interaction with the ACE-2 receptor, confirming S1 subunit as an attractive target for vaccinations or antiviral therapy (8).

## References:

1. Rota, P.A. *et al.* (2003) *Science* **300**:1394.
2. Bosch, B.J. *et al.* (2003). *J. Virol.* **77**:8801.
3. Belouzard, S. *et al.* (2009) *Proc. Natl. Acad. Sci. USA* **106**:5871.
4. Millet, J.K. and G. R. Whittaker (2015) *Virus Res.* **202**:120.
5. Li, W. *et al.* (2003) *Nature* **426**:450.
6. Wong, S.K. *et al.* (2004) *J. Biol. Chem.* **279**:3197.
7. Ortega, J.T. *et al.* (2020) *EXCLI J.* **19**:410.
8. Du, L. *et al.* (2009) *Nat. Rev. Microbiol.* **7**:226.