

DESCRIPTION

Source Chinese Hamster Ovary cell line, CHO-derived batcov ratg13 Spike protein
Val16-Pro12099 (Lys982Pro, Val983Pro), with a C-terminal 6-His tag
Accession # QHR63300.2

N-terminal Sequence Analysis Val 16

Predicted Molecular Mass 134 kDa

SPECIFICATIONS

SDS-PAGE 154-175 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.

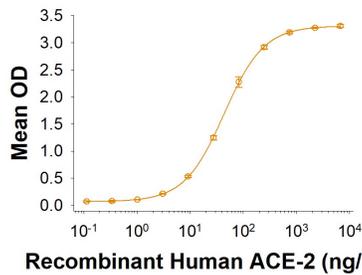
Shipping The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below.

Stability & Storage 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.

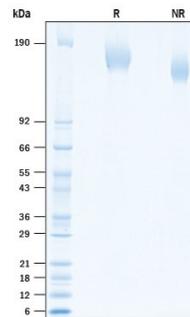
DATA

Binding Activity



Recombinant BatCoV RaTG13 Spike His-tag (Catalog # 10660-CV) binds Recombinant Human ACE-2 Fc Chimera (Catalog # 10544-ZN) in a functional ELISA.

SDS-PAGE



2 µg/lane of Recombinant BatCoV RaTG13 Spike His-tag (Catalog # 10660-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 154-175 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 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). Based on structural biology studies, the receptor binding domain (RBD), located in the C-terminal region of S1, can be oriented either in the up/standing or down/lying state (6). The standing state is associated with higher pathogenicity and both SARS-CoV-1 and MERS can access this state due to the flexibility in their respective RBDs. A similar two-state structure and flexibility is found in the SARS-Cov-2 RBD (7). SARS-Cov 2 is likely originated from Bat coronavirus RaTG13. Based on amino acid (aa) sequence homology, the Spike protein of RaTG13 shares 76% and 97% homology with Spike protein of SARS-CoV and SARS-CoV2, respectively. Despite high homology to SARS-COV2, five of the six key amino acids involved in ACE2 binding are different in RaTG13, leading to >1000 fold weaker binding to human ACE2 (8, 9). 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 (10). Polyclonal antibodies to the RBD of the SARS-CoV-2 protein have been shown to inhibit interaction with the ACE2 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).

References:

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