R&D SYSTEMS
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Recombinant SARS-CoV-2 B.1.1.529 Spike S1 Subunit His-tag

Catalog Number: 11070-CV

DESCRIPTION

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike S1 Subunit protein

SARS-CoV-2 B.1.1.529 Spike S1 (Val16-Pro681) (Ala67Val, His69del, Val70del, Thr95Ile, Gly142Asp, Val143del, Tyr144del, Tyr145del, Asn211del, Leu212Ile, ins214Glu-Pro-Glu, Gly339Asp, Ser371Leu, Ser373Pro, Ser375Phe, Lys417Asn, Asn440Lys, Gly446Ser, Ser477Asn, Thr478Lys, Glu484Ala, Gln493Arg, Gly496Ser, Gln498Arg, Asn501Tyr, Tyr505His, Thr547Lys, Asp614Gly, His655Tyr, Asn679Lys, Pro681His) Accession # YP_009724390.1	6-His tag
N-terminus	C-terminus

N-terminal Sequence Analysis	Val16
Predicted Molecular Mass	75 kDa

SPECIFICATIONS

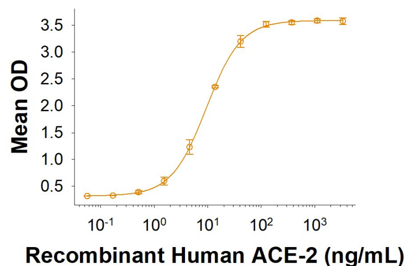
SDS-PAGE	104-116 kDa, under reducing conditions
Activity	Measured by its binding ability in a functional ELISA with Recombinant Human ACE-2 His-tag (Catalog # 933-ZN).
Endotoxin Level	<0.10 EU per 1 μg of the protein by the LAL method.
Purity	>95%, by SDS-PAGE visualized with Silver Staining and quantitative 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.

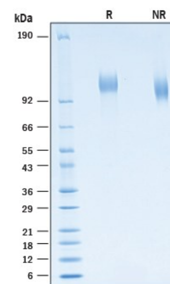
DATA

Binding Activity



Recombinant SARS-CoV-2 B.1.1.529 Spike S1 Subunit His-tag Protein Binding Activity. Recombinant SARS-CoV-2 B.1.1.529 Spike S1 Subunit His-tag (Catalog # 11070-CV) binds Recombinant Human ACE-2 His-tag (Catalog # 933-ZN) in a functional ELISA.

SDS-PAGE



Recombinant SARS-CoV-2 B.1.1.529 Spike S1 Subunit His-tag Protein SDS-PAGE. 2 μg/lane of Recombinant SARS-CoV-2 B.1.1.529 Spike S1 Subunit His-tag Protein (Catalog # 11070-CV) was resolved with SDS-PAGE under reducing (R) and non-reducing (NR) conditions and visualized by Coomassie® Blue staining, showing bands at 104-116 kDa.

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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). 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 difference 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). 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 29 mutations in the S1 subunit of SARS-Cov-2, of which 15 are located in the RBD domain (14-16). The majority of the RBD mutations are involved in ACE-2 binding and they allow the Omicron variant to bind ACE-2 with greater affinity and, potentially, increased transmissibility (14-18). Additionally, several of these mutations have been identified in facilitating immune escape and reducing neutralization activity to several monoclonal antibodies (14-16). The Omicron variant also contains four mutations within the N terminal domain of the S1 subunit, which may enhance immune evasion (16). Several unique mutations are present in the S1 subunit which have unknown impacts on receptor binding or antibody neutralization. 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 (19).

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