

Recombinant SARS-CoV-2 B.1.351 Spike (GCN4-IZ) His-tag Alexa Fluor® 488

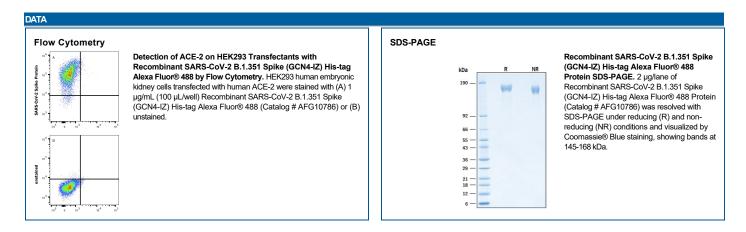
Catalog Number: AFG10786

Source	Human embryonic kidney cell, HEK293-derived sars-cov-2 Spike protein				
	SARS-CoV-2 B.1.351 Spike (Val16-Lys1211) (Asp80Ala, Asp215Gly, Leu242 del, Ala243 del, Leu244 del, Lys417Asn, Glu484Lys, Asn501Tyr, Asp614Gly, Ala701Val) (Arg682Ser, Arg685Ser, Lys986Pro, Val987Pro) Accession # YP_009724390.1	GCN4-IZ	6-His tag		
	N-terminus		C-termini		

N-terminal Sequence Analysis	Val16
Structure / Form	Labeled with Alexa Fluor® 488 via amines
	Excitation Wavelength: 488 nm
	Emission Wavelength: 515-545 nm
Predicted Molecular	138 kDa

SPECIFICATIONS		
SDS-PAGE	145-168 kDa under reducing conditions.	
Activity	Measured by flow cytometry for its ability to bind HEK293 human embryonic kidney cells transfected with human ACE-2 at 0.25-1.00 μg/mL (100 μL/well). Please Note: Optimal dilutions should be determined by each laboratory for each application.	
Endotoxin Level	<1.0 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	Supplied as a 0.2 µm filtered solution in PBS with BSA as a carrier protein. See Certificate of Analysis for details.	

PREPARATION AND STORAGE		
Shipping	The product is shipped with dry ice or equivalent. Upon receipt, store it immediately at the temperature recommended below.	
Stability & Storage	Protect from light. Use a manual defrost freezer and avoid repeated freeze-thaw cycles.	
	 6 months from date of receipt, -20 to -70 °C as supplied. 	
	 1 month, 2 to 8 °C under sterile conditions after opening. 	
	 3 months, -20 to -70 °C under sterile conditions after opening. 	



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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 the 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 S protein of SARS-CoV-2 shares 75% and 29% amino acid (aa) sequence identity with the S protein of SARS-CoV-1 and MERS, respectively. The S Protein of the SARS-CoV-2 virus, like the SARS-CoV-1 counterpart, binds Angiotensin-Converting Enzyme 2 (ACE-2), but with much higher affinity and faster binding kinetics through the receptor binding domain (RBD) located in the C-terminal region of S1 (6). Based on structural biology studies, the RBD can be oriented either in the up/standing or down/lying state with the up/standing state associated with higher pathogenicity (7). Polyclonal antibodies to the RBD of the SARS-CoV-2 protein have been shown to inhibit interaction with the ACE-2 receptor, confirming RBD as an attractive target for vaccinations or antiviral therapy (8). It has been demonstrated that the S Protein can invade host cells through the CD147/EMMPRIN receptor and mediate membrane fusion (9, 10). A SARS-CoV-2 variant carrying amino acid substitutions N501Y, K417N, and E484K in the RBD raised the most concerns. This B.1.351 lineage, also known and 501Y.V2 variant, was first identified in the Eastern Cape province of South Africa in December 2020 and spread quickly to become the most dominant strain in the second COVID wave in South Africa (11). Two of these mutations K417N and E484K locate at the receptor binding motif (RBM) and are not found in other variants (11). The N501Y mutation is also found in London (B.1.1.7 lineage) and Brazil (P.1 lineage). The B.1.351 lineage is reported to enter cells more easily due to its enhanced affinity to ACE-2 receptor (12). It is reported to reduce the efficacy of neutralizing antibody (12, 13).

References:

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