SARS-CoV-2 Spike RBD Alexa Fluor® 700-conjugated Antibody

Monoclonal Mouse IgG_1 Clone # 1056608

Catalog Number: FAB11227N

100 µg

DESCRIPTION

Species Reactivity	SARS-CoV-2
Specificity	It detects SARS-CoV-2 Omicron BA.1 in Direct ELISA. In ELISA, this antibody does not detect SARS-CoV-2 Alpha, Gamma or Delta variants.
Source	Monoclonal Mouse IgG_1 Clone # 1056608
Purification	Protein A or G purified from cell culture supernatant
Immunogen	Recombinant SARS-CoV-2 B.1.1.529 (Omicron) Spike RBD domain. Arg319-Phe541 Accession # YP_009724390.1
Conjugate	Alexa Fluor 700 Excitation Wavelength: 675-700 nm Emission Wavelength: 723 nm
Formulation	Supplied 0.2mg/ml in 1X PBS with RDF1 and 0.09% Sodium Azide *Contains <0.1% Sodium Azide, which is not hazardous at this concentration according to GHS classifications. Refer to the Safety Data Sheet (SDS) for additional information and handling instructions.

APPLICATIONS

Please Note: *Optimal dilutions should be determined by each laboratory for each application. General Protocols are available in the Technical Information section on our website.*

Blockade of Receptor-ligand Interaction	Optimal dilution of this antibody should be experimentally determined.
Immunocytochemistry	Optimal dilution of this antibody should be experimentally determined.

PREPARATION AND STORAGE

Shipping	The product is shipped with polar packs. Upon receipt, store it immediately at the temperature recommended below.
Stability & Storage	Protect from light. Do not freeze. 12 months from date of receipt, 2 to 8 °C as supplied

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). A receptor binding domain (RBD) in the C-terminus of the S1 subunit has been identified and the RBD of SARS-CoV-2 shares 73% amino acid (aa) identity with the RBD of the SARS-CoV-1, but only 22% aa identity with the RBD of MERS-CoV (6, 7). The low aa sequence homology is consistent with the finding that SARS and MERS-CoV bind different cellular receptors (8). The RBD of SARS-CoV-2 binds a metallopeptidase, angiotensin-converting enzyme 2 (ACE-2), similar to SARS-CoV-1, but with much higher affinity and faster binding kinetics (9). Before binding to the ACE-2 receptor, structural analysis of the S1 trimer shows that only one of the three RBD domains 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 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 (12). 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 15 mutations in RBD domain that potentially affect viral fitness and transmissibility. The majority of the mutations are involved in ACE-2 binding and Omicron binds ACE-2 with greater affinity, potentially explaining its increased transmissibility (13, 14). Several of these mutations are also identified in facilitating immune escape and reducing neutralization activity to several monoclonal antibodies (13). Additionally, a series of new mutations are present in the RBD which have unknown impacts on receptor binding or antibody neutralization.

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