

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ACE-2 in direct ELISAs.
Source	Monoclonal Mouse IgG _{2A} Clone # 1034734
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived human ACE-2 Gln18-Ser740 Accession # Q9BYF1
Conjugate	Alexa Fluor 647 Excitation Wavelength: 650 nm Emission Wavelength: 668 nm
Formulation	Supplied 0.2mg/mL in a saline solution containing BSA and 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.

Flow Cytometry	Titration recommended for optimal concentration with starting range of 0.1-1 µg/1 million cells. Sample used for this experiment was HEK293 Human Cell Line Transfected with Human ACE-2 and eGFP
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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

Angiotensin I Converting Enzyme (ACE-2), also called ACEH (ACE homologue), is a dimeric, zinc-dependent metalloprotease of the ACE family that also includes somatic and germinal ACE (1, 2). ACE-2 mRNA is found at high levels in heart, testis, and kidney and at lower levels in a wide variety of tissues (1, 3). ACE-2 is the SARS-CoV and SARS-CoV2 Spike protein receptor *in vivo* (4-6), functions catalytically as a carboxypeptidase to cleave several substrates including angiotensins I and II, and acts as a partner for B0AT1-family amino acid transporters (1, 2). Through these functions, ACE-2 has been shown to be involved in several diseases including SARS, COVID19, acute lung injury (4, 7), heart disease (8), liver and lung fibrosis (9), inflammatory lung disease (10), and cardiopulmonary disease (11). Full length ACE-2 protein includes an extracellular region composed of a single N-terminal peptidase domain and C-terminal collectrin-like domain (CLD), a transmembrane domain, and a short cytoplasmic tail (12). The N-terminal peptidase region is required for binding to SARS-CoV and SARSCoV2 spike proteins, while the CLD contains a region that promotes dimerization and association with amino acid transporters (2). The peptidase domain contains a long deep cleft that undergoes a large hinge-bending movement at substrate and inhibitor binding (12). Classical ACE inhibitors such as captopril and lisinopril do not inhibit ACE-2 activity and inhibitors of ACE-2 do not inhibit ACE activity (13).

References:

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