

DESCRIPTION

Species Reactivity	Human
Specificity	Detects human ACE-2 in direct ELISAs.
Source	Monoclonal Mouse IgG ₁ Clone # 1038216
Purification	Protein A or G purified from hybridoma culture supernatant
Immunogen	Mouse myeloma cell line NS0-derived human ACE-2 protein Gln18-Ser740 Accession # Q9BYF1
Formulation	Lyophilized from a 0.2 µm filtered solution in PBS with Trehalose. See Certificate of Analysis for details. *Small pack size (-SP) is supplied either lyophilized or as a 0.2 µm filtered solution in PBS.

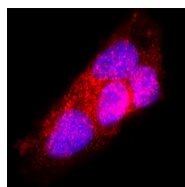
APPLICATIONS

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.

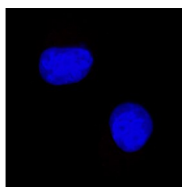
	Recommended Concentration	Sample
Immunocytochemistry	8-25 µg/mL	Immersion fixed HepG2 human hepatocellular carcinoma cell
Immunohistochemistry	5-25 µg/mL	Immersion fixed paraffin-embedded sections of human kidney

DATA

Immunocytochemistry



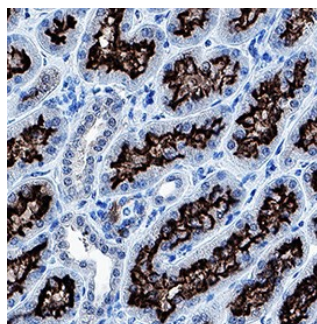
Positive (HepG2 cells)



Negative (MCF-7 cells)

ACE-2 in HepG2 Human Cell Line. ACE-2 was detected in immersion fixed HepG2 human hepatocellular carcinoma cell (positive staining) line and MCF-7 human breast cancer cell line (negative control) using Mouse Anti-Human ACE-2 Monoclonal Antibody (Catalog # MAB9335) at 8 µg/mL for 3 hours at room temperature. Cells were stained using the NorthernLights™ 557-conjugated Anti-Mouse IgG Secondary Antibody (red; Catalog # NL007) and counterstained with DAPI (blue). Specific staining was localized to cytoplasm. Staining was performed using our protocol for Fluorescent ICC Staining of Non-adherent Cells.

Immunohistochemistry



ACE-2 in Human Kidney. ACE-2 was detected in immersion fixed paraffin-embedded sections of human kidney using Mouse Anti-Human ACE-2 Monoclonal Antibody (Catalog # MAB9335) at 5 µg/mL for 1 hour at room temperature followed by incubation with the Anti-Mouse IgG VisUCyte™ HRP Polymer Antibody (Catalog # VC001). Before incubation with the primary antibody, tissue was subjected to heat-induced epitope retrieval using Antigen Retrieval Reagent-Basic (Catalog # CTS013). Tissue was stained using DAB (brown) and counterstained with hematoxylin (blue). Specific staining was localized to cell membrane in convoluted tubules. Staining was performed using our protocol for IHC Staining with VisUCyte HRP Polymer Detection Reagents.

PREPARATION AND STORAGE

Reconstitution	Reconstitute at 0.5 mg/mL in sterile PBS.
Shipping	The product is shipped at ambient temperature. Upon receipt, store it immediately at the temperature recommended below. *Small pack size (-SP) is shipped with polar packs. Upon receipt, store it immediately at -20 to -70 °C
Stability & Storage	Use a manual defrost freezer and avoid repeated freeze-thaw cycles. <ul style="list-style-type: none"> • 12 months from date of receipt, -20 to -70 °C as supplied. • 1 month, 2 to 8 °C under sterile conditions after reconstitution. • 6 months, -20 to -70 °C under sterile conditions after reconstitution.

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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