# Human ACE-2 Antibody

Recombinant Monoclonal Rabbit IgG Clone # 2817F
Catalog Number: MAB9337

## DESCRIPTION

| | |
|---|---|
| **Species Reactivity** | Human |
| **Specificity** | Detects human ACE-2 in direct ELISAs. |
| **Source** | Recombinant Monoclonal Rabbit IgG Clone # 2817F |
| **Purification** | Protein A or G purified from hybridoma culture supernatant |
| **Immunogen** | Mouse myeloma cell line NS0-derived human ACE-2<br>Gln18-Ser740<br>Accession # Q9BYF1 |
| **Formulation** | Lyophilized from a 0.2 μm filtered solution in PBS with Trehalose. See Certificate of Analysis for details.<br>*Small pack size (-SP) is supplied either lyophilized or as a 0.2 μm filtered solution in PBS. |

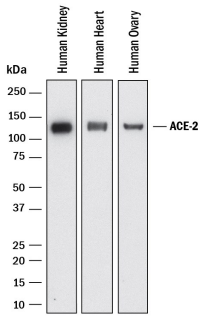
## APPLICATIONS

**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 |
|---|---|---|
| **Western Blot** | 1 µg/mL | Human kidney, human heart, and human ovary |
| **Simple Western** | 20 µg/mL | Human kidney, human heart, and human ovary |

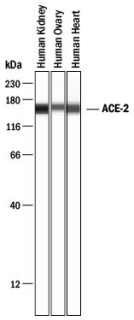
## DATA

### Western Blot




**Detection of Human ACE-2 by Western Blot.** Western blot shows lysates of human kidney, human heart, and human ovary. PVDF membrane was probed with 1 µg/mL of Rabbit Anti-Human ACE-2 Monoclonal Antibody (Catalog # MAB9337) followed by HRP-conjugated Anti-Rabbit IgG Secondary Antibody (Catalog # HAF008). A specific band was detected for ACE-2 at approximately 120 kDa (as indicated). This experiment was conducted under reducing conditions and using Western Blot Buffer Group 1.

### Simple Western




**Detection of Human ACE-2 by Simple Western™.** Simple Western lane view shows lysates of human kidney, human ovary and human heart, loaded at 0.2 mg/mL. A specific band was detected for ACE-2 at approximately 157 kDa (as indicated) using 20 µg/mL of Rabbit Anti-Human ACE-2 Monoclonal Antibody (Catalog # MAB9337) . This experiment was conducted under reducing conditions and using the 12-230 kDa separation system.

## 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.<br>*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.**<br>• 12 months from date of receipt, -20 to -70 °C as supplied.<br>• 1 month, 2 to 8 °C under sterile conditions after reconstitution.<br>• 6 months, -20 to -70 °C under sterile conditions after reconstitution. |



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## 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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