

**ACE Antibody (CT)**  
**Rabbit Polyclonal Antibody**  
**Catalog # ABV11253****Specification**

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**ACE Antibody (CT) - Product Information**

|                   |                        |
|-------------------|------------------------|
| Application       | WB                     |
| Primary Accession | <a href="#">P12821</a> |
| Reactivity        | Human, Mouse           |
| Host              | Rabbit                 |
| Clonality         | Polyclonal             |
| Isotype           | Rabbit IgG             |
| Calculated MW     | 149715                 |

**ACE Antibody (CT) - Additional Information****Gene ID** 1636

|                     |                                         |
|---------------------|-----------------------------------------|
| Positive Control    | Western blot: mouse liung tissue lysate |
| Application & Usage | Western blot: 1:1000                    |

**Other Names**

ACE; DCP; DCP1; Angiotensin-converting enzyme; Dipeptidyl carboxypeptidase I; Kininase II;  
CD\_antigen - CD143; Angiotensin-converting enzyme, soluble form

**Target/Specificity**

ACE

**Antibody Form**

Liquid

**Appearance**

Colorless liquid

**Formulation**

100 µl of antibody in PBS with 0.09% (W/V) sodium azide

**Handling**

The antibody solution should be gently mixed before use.

**Reconstitution & Storage**

-20 °C

**Background Descriptions****Precautions**

ACE Antibody (CT) is for research use only and not for use in diagnostic or therapeutic procedures.

**ACE Antibody (CT) - Protein Information**

**Name** ACE {ECO:0000303|PubMed:2849100, ECO:0000312|HGNC:HGNC:2707}

### Function

Dipeptidyl carboxypeptidase that removes dipeptides from the C-terminus of a variety of circulating hormones, such as angiotensin I, bradykinin or enkephalins, thereby playing a key role in the regulation of blood pressure, electrolyte homeostasis or synaptic plasticity (PubMed:<a href="http://www.uniprot.org/citations/2558109" target="\_blank">2558109</a>, PubMed:<a href="http://www.uniprot.org/citations/4322742" target="\_blank">4322742</a>, PubMed:<a href="http://www.uniprot.org/citations/7683654" target="\_blank">7683654</a>, PubMed:<a href="http://www.uniprot.org/citations/7523412" target="\_blank">7523412</a>, PubMed:<a href="http://www.uniprot.org/citations/15615692" target="\_blank">15615692</a>, PubMed:<a href="http://www.uniprot.org/citations/20826823" target="\_blank">20826823</a>). Composed of two similar catalytic domains, each possessing a functional active site, with different selectivity for substrates (PubMed:<a href="http://www.uniprot.org/citations/1851160" target="\_blank">1851160</a>, PubMed:<a href="http://www.uniprot.org/citations/1320019" target="\_blank">1320019</a>, PubMed:<a href="http://www.uniprot.org/citations/7683654" target="\_blank">7683654</a>, PubMed:<a href="http://www.uniprot.org/citations/7876104" target="\_blank">7876104</a>, PubMed:<a href="http://www.uniprot.org/citations/10913258" target="\_blank">10913258</a>, PubMed:<a href="http://www.uniprot.org/citations/19773553" target="\_blank">19773553</a>). Plays a major role in the angiotensin-renin system that regulates blood pressure and sodium retention by the kidney by converting angiotensin I to angiotensin II, resulting in an increase of the vasoconstrictor activity of angiotensin (PubMed:<a href="http://www.uniprot.org/citations/4322742" target="\_blank">4322742</a>, PubMed:<a href="http://www.uniprot.org/citations/1851160" target="\_blank">1851160</a>, PubMed:<a href="http://www.uniprot.org/citations/11432860" target="\_blank">11432860</a>, PubMed:<a href="http://www.uniprot.org/citations/19773553" target="\_blank">19773553</a>, PubMed:<a href="http://www.uniprot.org/citations/23056909" target="\_blank">23056909</a>). Also able to inactivate bradykinin, a potent vasodilator, and therefore enhance the blood pressure response (PubMed:<a href="http://www.uniprot.org/citations/2558109" target="\_blank">2558109</a>, PubMed:<a href="http://www.uniprot.org/citations/6055465" target="\_blank">6055465</a>, PubMed:<a href="http://www.uniprot.org/citations/4322742" target="\_blank">4322742</a>, PubMed:<a href="http://www.uniprot.org/citations/6270633" target="\_blank">6270633</a>, PubMed:<a href="http://www.uniprot.org/citations/7683654" target="\_blank">7683654</a>, PubMed:<a href="http://www.uniprot.org/citations/15615692" target="\_blank">15615692</a>). Acts as a regulator of synaptic transmission by mediating cleavage of neuropeptide hormones, such as substance P, neurotensin or enkephalins (PubMed:<a href="http://www.uniprot.org/citations/656131" target="\_blank">656131</a>, PubMed:<a href="http://www.uniprot.org/citations/6270633" target="\_blank">6270633</a>, PubMed:<a href="http://www.uniprot.org/citations/6208535" target="\_blank">6208535</a>, PubMed:<a href="http://www.uniprot.org/citations/15615692" target="\_blank">15615692</a>). Catalyzes degradation of different enkephalin neuropeptides (Met- enkephalin, Leu-enkephalin, Met-enkephalin-Arg-Phe and possibly Met- enkephalin-Arg-Gly-Leu) (PubMed:<a href="http://www.uniprot.org/citations/656131" target="\_blank">656131</a>, PubMed:<a href="http://www.uniprot.org/citations/6270633" target="\_blank">6270633</a>, PubMed:<a href="http://www.uniprot.org/citations/2982830" target="\_blank">2982830</a>). Acts as a regulator of synaptic plasticity in the nucleus accumbens of the brain by mediating cleavage of Met-enkephalin- Arg-Phe, a strong ligand of Mu-type opioid receptor OPRM1, into Met- enkephalin (By similarity). Met-enkephalin-Arg-Phe cleavage by ACE decreases activation of OPRM1, leading to long-term synaptic potentiation of glutamate release (By similarity). Also acts as a regulator of hematopoietic stem cell differentiation by mediating degradation of hemoregulatory peptide N-acetyl-SDKP (AcSDKP) (PubMed:<a href="http://www.uniprot.org/citations/8257427" target="\_blank">8257427</a>, PubMed:<a href="http://www.uniprot.org/citations/7876104" target="\_blank">7876104</a>, PubMed:<a href="http://www.uniprot.org/citations/8609242" target="\_blank">8609242</a>, PubMed:<a href="http://www.uniprot.org/citations/26403559" target="\_blank">26403559</a>). Acts as a regulator of cannabinoid signaling pathway by mediating degradation of hemopressin, an antagonist peptide of the cannabinoid receptor CNR1

(PubMed:<a href="http://www.uniprot.org/citations/18077343" target="\_blank">18077343</a>). Involved in amyloid-beta metabolism by catalyzing degradation of Amyloid-beta protein 40 and Amyloid-beta protein 42 peptides, thereby preventing plaque formation (PubMed:<a href="http://www.uniprot.org/citations/11604391" target="\_blank">11604391</a>, PubMed:<a href="http://www.uniprot.org/citations/16154999" target="\_blank">16154999</a>, PubMed:<a href="http://www.uniprot.org/citations/19773553" target="\_blank">19773553</a>). Catalyzes cleavage of cholecystokinin (maturation of Cholecystokinin-8 and Cholecystokinin-5) and Gonadoliberin-1 (both maturation and degradation) hormones (PubMed:<a href="http://www.uniprot.org/citations/2983326" target="\_blank">2983326</a>, PubMed:<a href="http://www.uniprot.org/citations/7683654" target="\_blank">7683654</a>, PubMed:<a href="http://www.uniprot.org/citations/9371719" target="\_blank">9371719</a>, PubMed:<a href="http://www.uniprot.org/citations/10336644" target="\_blank">10336644</a>). Degradation of hemoregulatory peptide N-acetyl-SDKP (AcSDKP) and amyloid-beta proteins is mediated by the N-terminal catalytic domain, while angiotensin I and cholecystokinin cleavage is mediated by the C-terminal catalytic region (PubMed:<a href="http://www.uniprot.org/citations/7876104" target="\_blank">7876104</a>, PubMed:<a href="http://www.uniprot.org/citations/10336644" target="\_blank">10336644</a>, PubMed:<a href="http://www.uniprot.org/citations/19773553" target="\_blank">19773553</a>).

### Cellular Location

Cell membrane; Single-pass type I membrane protein. Cytoplasm {ECO:0000250|UniProtKB:P09470}. Note=Detected in both cell membrane and cytoplasm in neurons. {ECO:0000250|UniProtKB:P09470} [Isoform Testis-specific]: Cell membrane; Single-pass type I membrane protein. Secreted. Note=The testis-specific isoform can be cleaved before the transmembrane region, releasing a soluble form

### Tissue Location

Ubiquitously expressed, with highest levels in lung, kidney, heart, gastrointestinal system and prostate

## ACE Antibody (CT) - Protocols

Provided below are standard protocols that you may find useful for product applications.

- [Western Blot](#)
- [Blocking Peptides](#)
- [Dot Blot](#)
- [Immunohistochemistry](#)
- [Immunofluorescence](#)
- [Immunoprecipitation](#)
- [Flow Cytometry](#)
- [Cell Culture](#)

## ACE Antibody (CT) - Images

### ACE Antibody (CT) - Background

Angiotensin-converting enzyme (ACE) is a carboxy-terminal dipeptidyl exo-peptidase that converts Angiotensin I to the potent vasopressive hormone, Angiotensin II. There are two isoforms of ACE, the pulmonary ACEP and the testicular ACET. ACEP is a glycoprotein expressed in vascular endothelial cells of the lung, liver, adrenal cortex, pancreas, kidney and spleen. The ACET isoform is expressed exclusively in adult testis by developing sperm cells, specifically, late pachytene spermatocytes. Additionally, ACE inactivates bradykinin, a vasodepressor peptide, and is involved in fluid/electrolyte homeostasis. Although it bears significant sequence homology to ACE, ACE2 shows a more restricted pattern of expression. ACE is expressed ubiquitously throughout the vasculature while ACE2 is expressed only in cardiac, renal and testicular cells.