

ACE Antibody (Ascites)

Mouse Monoclonal Antibody (Mab)
Catalog # AM2111a

Specification

ACE Antibody (Ascites) - Product Information

Application WB,E Primary Accession P12821 Other Accession NP 000780 Reactivity Mouse Host Mouse Clonality **Monoclonal** Isotype IgG2a Calculated MW 149715 Antigen Region 1274-1306

ACE Antibody (Ascites) - Additional Information

Gene ID 1636

Other Names

Angiotensin-converting enzyme, ACE, 321-, Dipeptidyl carboxypeptidase I, Kininase II, CD143, Angiotensin-converting enzyme, soluble form, ACE, DCP, DCP1

Target/Specificity

This ACE antibody is generated from mice immunized with a KLH conjugated synthetic peptide between 1274-1306 amino acids from human ACE.

Dilution

WB~~1:500~16000

E~~Use at an assay dependent concentration.

Format

Mouse monoclonal antibody supplied in crude ascites with 0.09% (W/V) sodium azide.

Storage

Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

Precautions

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

ACE Antibody (Ascites) - 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:15615692, PubMed:20826823, PubMed:2558109, PubMed:4322742, PubMed:7523412, PubMed:7683654). Composed of two similar catalytic domains, each possessing a functional active site, with different selectivity for substrates (PubMed: 10913258, PubMed: 1320019, PubMed: 1851160, PubMed: 19773553, PubMed: 7683654, PubMed: 7876104). 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: 11432860, PubMed: 1851160, PubMed: 19773553, PubMed: 23056909, PubMed: 4322742). Also able to inactivate bradykinin, a potent vasodilator, and therefore enhance the blood pressure response (PubMed: 15615692, PubMed: 2558109, PubMed: 4322742, PubMed: 6055465, PubMed: 6270633, PubMed: 7683654). Acts as a regulator of synaptic transmission by mediating cleavage of neuropeptide hormones, such as substance P, neurotensin or enkephalins (PubMed:15615692, PubMed:6208535, PubMed:6270633, PubMed:656131), Catalyzes degradation of different enkephalin neuropeptides (Met- enkephalin, Leu-enkephalin, Met-enkephalin-Arg-Phe and possibly Met- enkephalin-Arg-Gly-Leu) (PubMed: 2982830, PubMed: 6270633, PubMed: 656131). 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: 26403559, PubMed: 7876104, PubMed: 8257427, PubMed: 8609242). Acts as a regulator of cannabinoid signaling pathway by mediating degradation of hemopressin, an antagonist peptide of the cannabinoid receptor CNR1 (PubMed: 18077343). Involved in amyloid-beta metabolism by catalyzing degradation of Amyloid-beta protein 40 and Amyloid-beta protein 42 peptides, thereby preventing plaque formation (PubMed: 11604391, PubMed: 16154999, PubMed: 19773553). Catalyzes cleavage of cholecystokinin (maturation of Cholecystokinin-8 and Cholecystokinin-5) and Gonadoliberin-1 (both maturation and degradation) hormones (PubMed: 10336644, PubMed: 2983326, PubMed: 7683654, PubMed: 9371719). 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: 10336644, PubMed: 19773553, PubMed: 7876104).

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 (Ascites) - Protocols

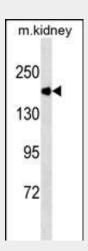
Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- <u>Immunohistochemistry</u>
- <u>Immunofluorescence</u>
- Immunoprecipitation



- Flow Cytomety
- Cell Culture

ACE Antibody (Ascites) - Images



ACE Antibody (Ascites)(Cat. #AM2111a) western blot analysis in mouse kidney tissue lysates (35µg/lane). This demonstrates the ACE antibody detected the ACE protein (arrow).

ACE Antibody (Ascites) - Background

This gene encodes an enzyme involved in catalyzing the conversion of angiotensin I into a physiologically active peptide angiotensin II. Angiotensin II is a potent vasopressor and aldosterone-stimulating peptide that controls blood pressure and fluid-electrolyte balance. This enzyme plays a key role in the renin-angiotensin system. Many studies have associated the presence or absence of a 287 bp Alu repeat element in this gene with the levels of circulating enzyme or cardiovascular pathophysiologies. Multiple alternatively spliced transcript variants encoding different isoforms have been identified, and two most abundant spliced variants encode the somatic form and the testicular form, respectively, that are equally active.

ACE Antibody (Ascites) - References

Dimitriou, G., et al. Pediatr. Pulmonol. 45(12):1233-1239(2010) Ince, D.A., et al. Genet Test Mol Biomarkers 14(5):643-647(2010) Procopciuc, L.M., et al. Eur. J. Intern. Med. 21(5):414-418(2010) Ash, G.I., et al. Med Sci Sports Exerc (2010) In press: Liu, L.W., et al. Chin. Med. J. 123(11):1382-1386(2010)