

ACE Antibody (C-term)

Purified Rabbit Polyclonal Antibody (Pab)

Catalog # AP7793b

Product Information

Application	WB, E
Primary Accession	P12821
Reactivity	Human, Mouse
Host	Rabbit
Clonality	Polyclonal
Isotype	Rabbit IgG
Clone Names	RB16489
Calculated MW	149715
Antigen Region	1274-1306

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 rabbits immunized with a KLH conjugated synthetic peptide between 1274-1306 amino acids from the C-terminal region of human ACE.
Dilution	WB~~1:1000 E~~Use at an assay dependent concentration.
Format	Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is prepared by Saturated Ammonium Sulfate (SAS) precipitation followed by dialysis against PBS.
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 (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

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

Background

ACE is 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 pathophysiology. Two most abundant alternatively spliced variants of this gene encode two isozymes - the somatic form and the testicular form that are equally active.

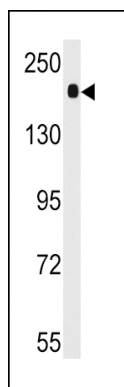
References

du Cheyron,D.,Crit. Care Med. 36 (12), 3178-3183 (2008)

Pang,S., Biochem. J. 358 (PT 1), 185-192 (2001)

Woodman,Z.L., Biochem. J. 347 PT 3, 711-718 (2000)

Images



Western blot analysis of anti-ACE Antibody (C-term)
(Cat.#AP7793b) in mouse lung tissue lysates (35ug/lane).
ACE (arrow) was detected using the purified Pab.

Please note: All products are 'FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC OR THERAPEUTIC PROCEDURES'.