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ACE Antibody (C-term) Blocking Peptide

Synthetic peptide
Catalog # BP7793b

Specification

ACE Antibody (C-term) Blocking Peptide - Product Information

Primary Accession P12821

ACE Antibody (C-term) Blocking Peptide - 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
The synthetic peptide sequence used to generate the antibody AP7793b was selected from the C-term region of human ACE. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

ACE Antibody (C-term) Blocking Peptide - 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:<a href="http://www.uniprot.org/citations/10913258"

target="_blank">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:<a href="http://www.uniprot.org/citations/10336644"

target="_blank">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 (C-term) Blocking Peptide - Protocols

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

- Blocking Peptides

ACE Antibody (C-term) Blocking Peptide - Images

ACE Antibody (C-term) Blocking Peptide - 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 pathophysiologies. Two most abundant alternatively spliced variants of this gene encode two isozymes - the somatic form and the testicular form that are equally active.

ACE Antibody (C-term) Blocking Peptide - 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)