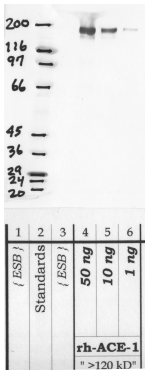
Anti-ACE Rabbit Polyclonal Antibody

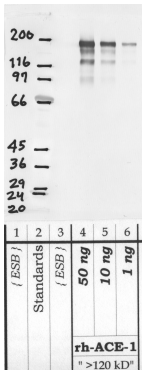
Angiotensin-converting enzyme, ACE, EC 3.4.15.1, Dipeptidyl carboxypeptidase I, Kininase II, CD antigen CD143, [Cleaved into: Angiotensin-converting enzyme, soluble form

Gene: ACE **UniProt:** P12821 **Host:** Rabbit **Clonality:** Polyclonal (pAb)

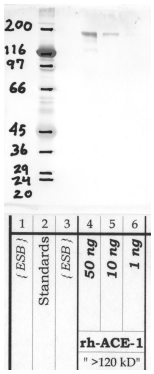
Product Image



Amino Catalytic domain
RP1ACE1



Carboxy Catalytic domain
RP3ACE1



Cytoplasmic domain
RP4ACE1

— Western Blot (3-panel validation across domain-specific antibody clones)

Actual Western blot validation for anti-ACE rabbit polyclonal antibody. 3-panel validation across domain-specific antibody clones — each panel labeled with its catalog number and target domain. Reducing conditions; 10–20 µg/lane on 7.5–10% SDS-PAGE.

Key Product Facts

Supplier	Triple Point Biologics, Inc. (Sudbury, Ontario, Canada)
Product Name	Anti-ACE Rabbit Polyclonal Antibody
Gene Symbol	ACE
Alternative Names	Angiotensin-converting enzyme, ACE, EC 3.4.15.1, Dipeptidyl carboxypeptidase I, Kininase II, CD antigen CD143, [Cleaved into: Angiotensin-converting enzyme, soluble form
UniProt ID	P12821
Host Species	Rabbit
Clonality	Polyclonal (pAb)
Isotype	IgG
Concentration	1mg/ml
Purity	Affinity purified
Pack Size	100ug
Validated Application	WB
Recommended Dilution	WB-1/1000
Species Reactivity	Validated- Human Potential-Mouse, Rat, Pan, Dog, Pig
Immunogen Type	Synthetic Peptide from Uniprot Sequence
Conjugation	none
Buffer / Preservatives	Sodium Chloride 500 mM, Sodium Phosphate 10 mM, Potassium Phosphate 1.8 mM, Potassium Chloride 2.7 mM, Glycerol 50% (v/v)
Website	triplepointbiologics.com/anti-ace-1-rabbit-polyclonal-antibody

Available Domain-Specific Variants

Triple Point Biologics produces antibodies to defined structural domains of ACE. Each variant is directed at a specific structural region of the protein. Exact peptide sequences are proprietary; each variant is validated for the applications listed.

Domain / Variant	Epitope Region (approx. residues)	Cat. No.	Availability

Amino Catalytic domain	**40–624** Region spanning the N-terminal (amino) catalytic domain of somatic human ACE, encompassing the HEXXH zinc-binding motif of the N-domain active site, the N-domain chloride-binding residues, and the surrounding α-helical bundle; lies upstream of the interdomain linker and C-domain	RP1ACE1	In catalog
Carboxy Catalytic domain	**40–624** Region spanning the C-terminal (carboxy) catalytic domain of somatic human ACE, containing the second HEXXH zinc-coordination motif, the C-domain chloride-binding site, and the surrounding helical scaffold; positioned downstream of the interdomain linker and upstream of the stalk/transmembrane segment	RP2ACE1	In catalog
Cytoplasmic domain	**1278–1306** Region corresponding to the short intracellular C-terminal tail of human somatic ACE, located immediately downstream of the single-pass transmembrane anchor; this cytoplasmic segment contains phosphorylatable residues implicated in ADAM17-mediated ectodomain shedding signalling	RP4ACE1	In catalog
—	**30–1306** Broad region encompassing both the N-domain and C-domain catalytic regions of the human somatic ACE ectodomain, suitable for detection of total ACE protein across membrane-bound and soluble forms without domain selectivity	RP-ACE1	In catalog
Carboxy Catalytic domain	Carboxy Catalytic domain	RP3ACE1	In catalog

Applications & Recommended Dilutions

App.	Species	Dilution Range	Notes
WB	Validated-Human Potential-Mouse, Rat, Pan, Dog, Pig	1:1000	Optimal dilution should be determined per laboratory and lot.

Optimal dilutions should be determined by each laboratory for each application. Triple Point Biologics antibodies include a peptide-neutralized negative control aliquot for specific-signal confirmation.

Storage & Handling

Short-term Storage	+4°C (1–2 weeks)
Long-term Storage	Aliquot and store at -20°C
Shipping Conditions	Shipped in styrofoam cooler on blue ice
Hazard Warning	Product may contain Sodium Azide — POISONOUS AND HAZARDOUS. Handle by trained personnel only.
Shelf Life	12 months from date of receipt under recommended conditions

Target Background

Enzyme Family & General Biology

Angiotensin-converting enzyme (ACE, EC 3.4.15.1; CD143) is a zinc-dependent dipeptidyl carboxypeptidase belonging to the gluzincin metallopeptidase family. The somatic isoform (1,306 amino acids, UniProt P12821) is a type I integral membrane glycoprotein that carries two tandem catalytic domains — the N-domain and C-domain — each harbouring a canonical HEXXH zinc-binding motif. A shorter testicular isoform (tACE) is transcribed from an internal promoter and encodes only the C-domain. Both isoforms are shed from the cell surface by ADAM17-mediated ectodomain shedding, generating catalytically active soluble forms detectable in plasma.

Expression & Localisation

ACE is most abundantly expressed on the luminal surface of vascular endothelial cells throughout the pulmonary circulation, where it is ideally positioned to process circulating peptide substrates in a single pass. High expression is also observed in kidney proximal tubule brush-border membranes, intestinal epithelium, choroid plexus, and testicular Leydig cells. In the immune compartment, ACE is expressed on monocyte-derived macrophages, dendritic cells, and a subset of T lymphocytes. The membrane-anchored form (CD143) is widely used as an endothelial surface marker. Soluble ACE in blood originates predominantly from endothelial shedding and shows measurable inter-individual variation partly governed by an insertion/deletion (I/D) polymorphism in intron 16.

Activation Mechanism & Chloride Dependence

ACE does not require zymogen processing for activity; both N- and C-domains are constitutively active in the mature protein. Catalysis follows a general acid–base mechanism in which the active-site zinc coordinates substrate carbonyl oxygen, facilitating nucleophilic attack by a zinc-bound water molecule. Crucially, both domains are allosterically stimulated by chloride ions, though with markedly different chloride concentration dependencies: the C-domain achieves half-maximal activation at low millimolar chloride, whereas the N-domain requires substantially higher chloride concentrations. This differential chloride sensitivity contributes to distinct substrate selectivities between the two domains under physiological ionic conditions.

Key Substrates & Biological Activities

The best-characterised ACE reaction is the conversion of angiotensin I (Ang I) to the potent vasopressor angiotensin II (Ang II) by removal of the C-terminal His-Leu dipeptide — a reaction carried out predominantly by the C-domain under physiological chloride. The C-domain also degrades bradykinin (a vasodilator) with high efficiency. The N-domain preferentially cleaves the haemoregulatory tetrapeptide AcSDKP (N-acetyl-Ser-Asp-Lys-Pro), enkephalins, neurotensin, substance P, and the amyloid-β peptide Aβ1-42. ACE additionally processes gonadotropin-releasing hormone (GnRH) and luteinizing hormone-releasing hormone (LHRH), implicating it in reproductive endocrinology.

Inhibitor Profile

ACE inhibitors (ACEi) constitute one of the most prescribed drug classes in cardiovascular medicine. First-generation compounds (captopril) are competitive, active-site-directed inhibitors that coordinate the catalytic zinc via a thiol group. Later

generations (enalaprilat, lisinopril, ramiprilat) employ carboxylate or phosphonate zinc-binding groups. Most clinical ACEi are non-selective across the two domains, though domain-selective inhibitors have been developed as research tools — notably RXP407 (N-domain selective) and RXPA380 (C-domain selective). Natural inhibitory peptides derived from dietary protein hydrolysates (e.g., casein-derived Ile-Pro-Pro) also demonstrate ACE inhibitory activity in vitro.

Disease Relevance

ACE occupies a central node in the renin–angiotensin–aldosterone system (RAAS) and is a principal therapeutic target in hypertension, heart failure, diabetic nephropathy, and chronic kidney disease. Elevated serum ACE activity is a diagnostic marker for sarcoidosis and is used clinically to monitor disease activity. Gain-of-function associations with the D-allele of the I/D polymorphism have been linked to increased cardiovascular and renal risk in some populations. Beyond cardiovascular disease, ACE dysregulation has been reported in Alzheimer's disease, COVID-19 pathophysiology (where ACE2, a paralogue, serves as the SARS-CoV-2 receptor), and male infertility — reflecting the broad physiological reach of this enzyme.

Gene & Protein Reference

Gene Symbol	ACE
HGNC	2707
NCBI Gene ID (Human)	1636
NCBI Gene ID (Mouse)	11421
Ensembl	ENSG00000159640
OMIM	106180
UniProt (Human)	P12821
UniProt (Mouse)	Q9Z1X5
Protein Family	Gluzincin metallopeptidase family; Peptidase M2 subfamily
Predicted MW	~150 kDa (polypeptide backbone ~149.8 kDa; heavily N-glycosylated)
Observed MW	~170–200 kDa (SDS-PAGE; glycosylation-dependent)
Chromosome	17q23.3
Paralogue	ACE2 (angiotensin-converting enzyme 2; UniProt Q9BYF1)
Key Substrates	Angiotensin I → Angiotensin II; Bradykinin (inactivation); AcSDKP; Enkephalins; Substance P; Neurotensin; Amyloid-β (Aβ1-42); GnRH/LHRH
Activated by	Chloride ions (allosteric activation, concentration-dependent and domain-specific); zinc cofactor (catalytic)
Inhibited by	Captopril, enalaprilat, lisinopril, ramiprilat, perindoprilat (clinical ACEi); RXP407 (N-domain selective, research tool); RXPA380 (C-domain selective, research tool); dietary casein-derived peptides (e.g., Ile-Pro-Pro) in vitro

About Triple Point Biologics

With over 30 years dedicated to antibody production, Triple Point Biologics is a Canadian company and the premier resource for proteinase and proteinase inhibitor research. Products have been cited in thousands of peer-reviewed publications.

The domain-specific antibody strategy — available in standard, ATK, SPA, and GSK kit formats — delivers predefined epitopes, built-in peptide-neutralized negative controls, and superior specificity for Western blot.

Website: www.triplepointbiologics.com

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