Anti-ADAM17 Rabbit Polyclonal Antibody

Disintegrin and metalloproteinase domain-containing protein 17, ADAM 17, EC 3.4.24.86, Snake venom-like protease, TNF-alpha convertase, TNF-alpha-converting enzyme, CD antigen CD156b,

Gene: ADAM17 **UniProt:** P78536 **Host:** Rabbit **Clonality:** Polyclonal (pAb)

Product Image

Image placeholder — Western Blot

— Western Blot

Suggested image: Western blot of relevant cell or tissue lysates under reducing conditions, probed with the domain-specific antibody. Include a positive-control lane and empty-vector or knockout negative control. Loading: 10–20 µg/lane on 7.5–10% SDS-PAGE.

Key Product Facts

Supplier	Triple Point Biologics, Inc. (Sudbury, Ontario, Canada)
Product Name	Anti-ADAM17 Rabbit Polyclonal Antibody
Gene Symbol	ADAM17
Alternative Names	Disintegrin and metalloproteinase domain-containing protein 17, ADAM 17, EC 3.4.24.86, Snake venom-like protease, TNF-alpha convertase, TNF-alpha-converting enzyme, CD antigen CD156b,
UniProt ID	P78536
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, 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-adam-17-rabbit-polyclonal-antibody

Available Domain-Specific Variants

Triple Point Biologics produces antibodies to defined structural domains of ADAM17. 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

Propeptide domain	**18–214** Region within the N-terminal prodomain of human ADAM17, which maintains zymogen latency via a cysteine-switch interaction with the catalytic zinc; this domain is removed by furin-family convertases in the trans-Golgi network during biosynthetic activation	RP2ADAM17	In catalog
Cytoplasmic domain	**693–824** Region within the short C-terminal cytoplasmic tail of human ADAM17, located downstream of the single-pass transmembrane helix; this intracellular segment contains phosphorylation sites implicated in signal-dependent regulation of sheddase activity	RP3ADAM17	In catalog
Activation site	**209–217** Region spanning the junction between the prodomain and catalytic domain of human ADAM17, encompassing the furin consensus cleavage site at which proprotein convertases remove the prodomain to generate the catalytically competent mature enzyme	RP4ADAM17	In catalog
Amino end of catalytic domain	**223–263** Region at the N-terminal portion of the metalloprotease catalytic domain of human ADAM17, proximal to the conserved HEXXH zinc-binding motif; this segment contributes to the substrate-binding groove and is critical for catalytic function	RP5ADAM17	In catalog
—	**215–824** Broad-coverage immunogen spanning multiple domains of human ADAM17, including elements of the prodomain, catalytic domain, disintegrin-like domain, cysteine-rich domain, and EGF-like region; suitable for general detection applications where domain-specific epitope mapping is not required	RP-ADAM17	In catalog

Applications & Recommended Dilutions

App.	Species	Dilution Range	Notes
WB	Validated-Human Potential-Mouse, Rat, 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

Family & General Biology

ADAM17 encodes a type I transmembrane zinc-dependent metalloprotease belonging to the ADAM (a disintegrin and metalloproteinase) family. ADAMs share a conserved multi-domain architecture — prodomain, metalloprotease, disintegrin-like, cysteine-rich, EGF-like, transmembrane, and cytoplasmic domains — and regulate cell-cell and cell-matrix communication principally through ectodomain shedding. ADAM17 was originally identified as the TNF-alpha converting enzyme (TACE) and remains one of the most extensively characterised sheddases in the family. Its closest paralogue, ADAM10, shares overlapping but distinct substrate specificity. ADAM17 is classified under EC 3.4.24.86 and is listed in MEROPS as metallopeptidase M12.217.

Expression & Localisation

ADAM17 is ubiquitously expressed across human tissues, with particularly high levels detected in heart, skeletal muscle, kidney, and immune cells including monocytes, macrophages, and T lymphocytes. The protein resides predominantly at the plasma membrane, oriented with its catalytic ectodomain facing the extracellular space. A pool of ADAM17 is also retained intracellularly in the Golgi and trans-Golgi network, where it undergoes prodomain removal during biosynthetic maturation. Surface expression levels are dynamically regulated by iRhom proteins (iRhom1 and iRhom2), which are required for ADAM17 trafficking and stability at the cell surface.

Activation Mechanism

ADAM17 is synthesised as a latent zymogen; the N-terminal prodomain maintains latency by coordinating the active-site zinc through a conserved cysteine residue (the cysteine-switch mechanism). Prodomain removal occurs in the trans-Golgi network by furin-family proprotein convertases, generating the mature, active form. Full catalytic activity at the cell surface is further regulated by iRhom1/2 adaptor proteins, cell-surface phosphatidylserine exposure, and integrin engagement. Stimuli including phorbol esters, calcium ionophores, growth factors, and inflammatory cytokines acutely increase ADAM17 shedding activity, in part through MAP-kinase-dependent pathways and conformational changes in the membrane-proximal domain.

Key Substrates

ADAM17 mediates ectodomain shedding of a remarkably broad substrate repertoire. Founding substrates include pro-TNF-alpha and pro-TGF-alpha; additional well-validated targets encompass IL-6 receptor alpha (IL-6Rα, enabling trans-signalling), L-selectin (SELL), HER2/ERBB2, HER4/ERBB4, epiregulin, amphiregulin, betacellulin, Notch ligands, and amyloid precursor protein (APP). ADAM17 also sheds angiotensin-converting enzyme 2 (ACE2) — the cellular receptor for SARS-CoV-2 — and platelet glycoprotein Ibα (GP1BA), implicating it in thrombosis. This breadth of substrate coverage positions ADAM17 as a central node in growth factor and cytokine signalling networks.

Disease Relevance

Dysregulated ADAM17 activity is documented across multiple pathological contexts. In chronic inflammatory disease, elevated ADAM17-mediated TNF-alpha and IL-6R shedding amplifies pro-inflammatory signalling; the enzyme is therefore of interest in

rheumatoid arthritis, inflammatory bowel disease, and sepsis. In oncology, ADAM17-dependent ERBB ligand and receptor shedding promotes tumour cell proliferation, invasion, and resistance to targeted therapy — overexpression has been reported in colorectal, breast, lung, and ovarian cancers. Cardiovascular studies implicate ADAM17 in cardiac hypertrophy, heart failure, and vascular remodelling. Genetic association with susceptibility to infectious and autoimmune disease has also been reported.

Inhibitor Profile

Endogenous inhibition of ADAM17 is provided by tissue inhibitor of metalloproteinases 3 (TIMP3), the only TIMP family member that potently inhibits ADAM-family sheddases at the cell surface; TIMP1 and TIMP2 are comparatively poor inhibitors of ADAM17. Synthetic hydroxamate-based broad-spectrum MMP/ADAM inhibitors (e.g., TAPI compounds, marimastat) block ADAM17 activity in vitro and in vivo. More selective small-molecule and antibody-based ADAM17 inhibitors have been developed as therapeutic candidates, exploiting the membrane-proximal regulatory region and substrate-binding exosites to achieve selectivity over structurally related family members.

Gene & Protein Reference

Gene Symbol	ADAM17
HGNC	HGNC:195
NCBI Gene ID (Human)	6868
NCBI Gene ID (Mouse)	11491
Ensembl	ENSG00000151694
OMIM	603639
UniProt (Human)	P78536
UniProt (Mouse)	Q9Z1N5
Protein Family	ADAM (a disintegrin and metalloproteinase) family; MEROPS M12.217
Predicted MW	~92 kDa (mature ectodomain form ~85 kDa after prodomain removal)
Observed MW	~100–130 kDa by SDS-PAGE (glycosylated full-length); mature shed form ~85 kDa
Chromosome	2p25.1
Paralogue	ADAM10 (closest paralogue; shares ectodomain shedding activity with overlapping substrate specificity)
Key Substrates	Pro-TNF-alpha, TGF-alpha, IL-6Rα, L-selectin (SELL), HER2/ERBB2, HER4/ERBB4, epiregulin, amphiregulin, betacellulin, APP, ACE2, GP1BA, Notch ligands
Activated by	Phorbol esters (PMA), calcium ionophores, growth factors, pro-inflammatory stimuli; iRhom1/iRhom2 chaperones required for surface trafficking; MAP kinase (ERK, p38) signalling; phosphatidylserine exposure
Inhibited by	TIMP3 (endogenous, potent); hydroxamate-class MMP/ADAM inhibitors (TAPI-0, TAPI-2, marimastat); selective anti-ADAM17 antibodies targeting the membrane-proximal domain

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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