Anti-ADAM12 Rabbit Polyclonal Antibody

Disintegrin and metalloproteinase domain-containing protein 12, ADAM 12, EC 3.4.24.-, Meltrin-alpha,

Gene: ADAM12 **UniProt:** O43184 **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-ADAM12 Rabbit Polyclonal Antibody
Gene Symbol	ADAM12
Alternative Names	Disintegrin and metalloproteinase domain-containing protein 12, ADAM 12, EC 3.4.24.-, Meltrin-alpha,
UniProt ID	O43184
Host Species	Rabbit
Clonality	Polyclonal (pAb)
Isotype	IgG
Concentration	1mg/ml
Purity	Affinity purified
Pack Size	100ug
Validated Application	WB, IHC
Recommended Dilution	WB-1/1000
Species Reactivity	Validated- Human Potential-Mouse, Pan, Monkey, Dog
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-12-rabbit-polyclonal-antibody

Available Domain-Specific Variants

Triple Point Biologics produces antibodies to defined structural domains of ADAM12. 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

Aminoterminal End	**208–258** Region within the N-terminal portion of the ADAM-12 prodomain, upstream of the furin cleavage site, encompassing the segment that coordinates the catalytic zinc via the cysteine-switch and maintains zymogen latency prior to trans-Golgi processing	RP1ADAM12	In catalog
Cytoplasmic domain ADAM-12 (long form)	**730–909** Region within the intracellular C-terminal cytoplasmic tail of the long membrane-anchored ADAM-12L isoform, distal to the single-pass transmembrane anchor, a segment absent from the secreted ADAM-12S splice variant and implicated in intracellular signalling and endocytic trafficking interactions	RP2ADAM12	In catalog
Amino end of Catalytic domain	**214–254** Region spanning the N-terminal portion of the metalloproteinase catalytic domain of ADAM-12, proximal to the prodomain cleavage site and upstream of the conserved HExxHxxGxxH zinc-binding motif, within the segment that forms part of the active-site cleft after furin-mediated activation	RP3ADAM12	In catalog
—	**208–909** Broad region spanning multiple ectodomains of ADAM-12, encompassing portions of the metalloproteinase, disintegrin, and cysteine-rich domains, providing recognition of the mature, processed ectodomain shared between both the long membrane-anchored and short secreted isoforms	RP-ADAM12	In catalog

Applications & Recommended Dilutions

App.	Species	Dilution Range	Notes
WB	Validated-Human Potential-Mouse, Pan, Monkey, Dog	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

ADAM-12 (meltrin-alpha) is a member of the ADAM (a disintegrin and metalloproteinase) family, a group of transmembrane and secreted zinc-dependent metalloproteinases that combine proteolytic activity with cell-adhesion capacity. The family is defined by a conserved domain architecture: prodomain, metalloproteinase domain, disintegrin domain, cysteine-rich domain, EGF-like domain, transmembrane anchor, and cytoplasmic tail. ADAM-12 exists as two splice variants — a long, membrane-anchored form (ADAM-12L) and a short, secreted form (ADAM-12S) — each with distinct tissue distributions and substrate repertoires.

Expression & Localisation

ADAM-12 expression is tightly regulated in a developmental and tissue-specific manner. It is highly expressed in skeletal muscle and placenta, with lower levels detected in heart, brain, and kidney. In adult tissues, basal expression is generally low but is markedly induced during muscle regeneration, adipogenesis, and in response to hypoxia. At the cellular level, ADAM-12 localises primarily to the plasma membrane, where the long isoform is anchored, while the secreted short isoform is shed into the extracellular space. Subcellular trafficking through the trans-Golgi network is required for prodomain removal and enzyme maturation.

Activation Mechanism

Like most ADAMs, ADAM-12 is synthesised as a zymogen; the N-terminal prodomain maintains latency by coordinating the catalytic zinc ion via a conserved cysteine switch mechanism. Activation occurs through furin-mediated cleavage of the prodomain in the trans-Golgi network or at the cell surface, releasing a catalytically competent ectodomain. The disintegrin loop and cysteine-rich domain are retained after activation and mediate integrin binding and substrate recruitment, respectively, thereby coupling proteolytic activity to cell-adhesion signalling.

Key Substrates & Proteolytic Functions

ADAM-12 cleaves a range of extracellular and membrane-anchored substrates relevant to growth factor signalling and matrix remodelling. Established ectodomain-shedding substrates include heparin-binding EGF-like growth factor (HB-EGF), IGFBP-3, and IGFBP-5, the latter two releasing IGF ligands to promote mitogenic signalling. ADAM-12 also processes delta-like ligand 1 (DLL1), modulating Notch pathway activity, and has been shown to shed several syndecans. Through these cleavage events, ADAM-12 regulates growth factor bioavailability and receptor transactivation independently of direct receptor binding.

Role in Muscle Biology & Cell Fusion

ADAM-12 was originally identified as a mediator of myoblast fusion during skeletal muscle differentiation; its expression peaks at the onset of myotube formation and declines in mature muscle. Genetic loss-of-function studies in mice demonstrate impaired myoblast fusion and reduced muscle fibre size, consistent with a required role in membrane merger. ADAM-12 is similarly induced during macrophage fusion to form multinucleated giant cells and during osteoclastogenesis from monocyte precursors, suggesting a conserved function in cell-cell fusion programmes across lineages.

Disease Relevance

Dysregulated ADAM-12 expression is associated with multiple pathological contexts. In cancer, ADAM-12 is upregulated in breast, gastric, hepatocellular, and bladder carcinomas, where elevated levels correlate with tumour invasion, metastasis, and poor prognosis. HIF-1α-driven transcription links ADAM-12 induction to hypoxic tumour microenvironments, and its shedding of IGFBPs promotes IGF-1R-dependent survival signalling. In fibrotic disease, ADAM-12 marks a subset of stromal fibroblasts and is elevated in cardiac and hepatic fibrosis. Its tractability as a biomarker in pregnancy-associated conditions, including pre-eclampsia, has also been explored.

Gene & Protein Reference

Gene Symbol	ADAM12
HGNC	HGNC:190
NCBI Gene ID (Human)	8038
NCBI Gene ID (Mouse)	27279
Ensembl	ENSG00000148848
OMIM	602714
UniProt (Human)	O43184
UniProt (Mouse)	Q9WTN0
Protein Family	ADAM (a disintegrin and metalloproteinase) family; meltrin subfamily
Predicted MW	~120 kDa (long form, ADAM-12L); ~90 kDa (short secreted form, ADAM-12S)
Observed MW	~120–130 kDa (full-length, glycosylated); ~90 kDa (secreted isoform) by SDS-PAGE
Chromosome	10q26.2
Paralogue	ADAM19 (meltrin-beta); also related to ADAM9 within the meltrin/ADAM subgroup
Key Substrates	HB-EGF, IGFBP-3, IGFBP-5, DLL1 (Delta-like ligand 1), syndecan-4
Activated by	Furin-mediated prodomain cleavage (cysteine-switch mechanism); HIF-1α-driven transcriptional upregulation under hypoxia
Inhibited by	TIMP-3 (primary endogenous inhibitor of the ectodomain); hydroxamate-based broad-spectrum metalloproteinase inhibitors (e.g. batimastat, marimastat)

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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Product specifications subject to change. Contact Triple Point Biologics for current product and availability information.