

Annexin A1 Antibody (C-term) Blocking Peptide
Synthetic peptide
Catalog # BP6584b**Specification**

Annexin A1 Antibody (C-term) Blocking Peptide - Product InformationPrimary Accession [P04083](#)**Annexin A1 Antibody (C-term) Blocking Peptide - Additional Information****Gene ID** 301**Other Names**

Annexin A1, Annexin I, Annexin-1, Calpactin II, Calpactin-2, Chromobindin-9, Lipocortin I, Phospholipase A2 inhibitory protein, p35, ANXA1, ANX1, LPC1

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP6584b](/products/AP6584b) was selected from the C-term region of human Annexin A1. 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.

Annexin A1 Antibody (C-term) Blocking Peptide - Protein Information**Name** ANXA1**Synonyms** ANX1, LPC1**Function**

Plays important roles in the innate immune response as effector of glucocorticoid-mediated responses and regulator of the inflammatory process. Has anti-inflammatory activity (PubMed: [8425544](http://www.uniprot.org/citations/8425544)). Plays a role in glucocorticoid-mediated down-regulation of the early phase of the inflammatory response (By similarity). Contributes to the adaptive immune response by enhancing signaling cascades that are triggered by T-cell activation, regulates differentiation and proliferation of activated T cells (PubMed: [17008549](http://www.uniprot.org/citations/17008549)). Promotes the differentiation of T cells into Th1 cells and negatively regulates differentiation into Th2 cells (PubMed: [17008549](http://www.uniprot.org/citations/17008549))

target="_blank">17008549). Has no effect on unstimulated T cells (PubMed:17008549). Negatively regulates hormone exocytosis via activation of the formyl peptide receptors and reorganization of the actin cytoskeleton (PubMed:19625660). Has high affinity for Ca(2+) and can bind up to eight Ca(2+) ions (By similarity). Displays Ca(2+)-dependent binding to phospholipid membranes (PubMed:2532504, PubMed:8557678). Plays a role in the formation of phagocytic cups and phagosomes. Plays a role in phagocytosis by mediating the Ca(2+)-dependent interaction between phagosomes and the actin cytoskeleton (By similarity). In the context of antitumor immunity, interacts with FPR1 on dendritic cells allowing for tumor-associated antigens uptake and cross-presentation to T cells to mount an antitumor specific T cell response.

Cellular Location

Nucleus. Cytoplasm. Cell projection, cilium {ECO:0000250|UniProtKB:P46193}. Cell membrane. Membrane; Peripheral membrane protein. Endosome membrane {ECO:0000250|UniProtKB:P07150}; Peripheral membrane protein {ECO:0000250|UniProtKB:P07150}. Basolateral cell membrane {ECO:0000250|UniProtKB:P51662}. Apical cell membrane {ECO:0000250|UniProtKB:P10107}. Lateral cell membrane {ECO:0000250|UniProtKB:P10107}. Secreted. Secreted, extracellular space. Cell membrane; Peripheral membrane protein; Extracellular side. Secreted, extracellular exosome. Cytoplasmic vesicle, secretory vesicle lumen. Cell projection, phagocytic cup {ECO:0000250|UniProtKB:P10107}. Early endosome {ECO:0000250|UniProtKB:P19619}. Cytoplasmic vesicle membrane {ECO:0000250|UniProtKB:P19619}; Peripheral membrane protein {ECO:0000250|UniProtKB:P19619}. Note=Secreted, at least in part via exosomes and other secretory vesicles. Detected in exosomes and other extracellular vesicles (PubMed:25664854). Alternatively, the secretion is dependent on protein unfolding and facilitated by the cargo receptor TMED10; it results in the protein translocation from the cytoplasm into ERGIC (endoplasmic reticulum-Golgi intermediate compartment) followed by vesicle entry and secretion (PubMed:32272059). Detected in gelatinase granules in resting neutrophils (PubMed:10772777). Secretion is increased in response to wounding and inflammation (PubMed:25664854). Secretion is increased upon T-cell activation (PubMed:17008549). Neutrophil adhesion to endothelial cells stimulates secretion via gelatinase granules, but foreign particle phagocytosis has no effect (PubMed:10772777). Colocalizes with actin fibers at phagocytic cups (By similarity). Displays calcium-dependent binding to phospholipid membranes (PubMed:2532504, PubMed:8557678) {ECO:0000250|UniProtKB:P10107, ECO:0000269|PubMed:10772777, ECO:0000269|PubMed:17008549, ECO:0000269|PubMed:2532504, ECO:0000269|PubMed:25664854, ECO:0000269|PubMed:32272059, ECO:0000269|PubMed:8557678}

Tissue Location

Detected in resting neutrophils (PubMed:10772777). Detected in peripheral blood T cells (PubMed:17008549). Detected in extracellular vesicles in blood serum from patients with inflammatory bowel disease, but not in serum from healthy donors (PubMed:25664854) Detected in placenta (at protein level) (PubMed:2532504). Detected in liver.

Annexin A1 Antibody (C-term) Blocking Peptide - Protocols

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

- [Blocking Peptides](#)

Annexin A1 Antibody (C-term) Blocking Peptide - Images

Annexin A1 Antibody (C-term) Blocking Peptide - Background

Annexin I belongs to a family of Ca^{2+} -dependent phospholipid binding proteins which have a molecular weight of approximately 35,000 to 40,000 and are preferentially located on the cytosolic face of the plasma membrane. Annexin I protein has an apparent relative molecular mass of 40 kDa, with phospholipase A2 inhibitory activity. Since phospholipase A2 is required for the biosynthesis of the potent mediators of inflammation, prostaglandins and leukotrienes, annexin I may have potential anti-inflammatory activity.

Annexin A1 Antibody (C-term) Blocking Peptide - References

Shimoji, T., J. Cell. Biochem. 106 (6), 1123-1135 (2009) Ang, E.Z., Mol. Cancer Res. 7 (2), 266-274 (2009)