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# CMKOR1 Antibody (C-term)

**Affinity Purified Rabbit Polyclonal Antibody (Pab)**
**Catalog # AP10486b**

## Specification

### CMKOR1 Antibody (C-term) - Product Information

| | |
|---|---|
| Application | **FC, WB,E** |
| Primary Accession | P25106 |
| Other Accession | NP_064707.1 |
| Reactivity | **Human** |
| Host | **Rabbit** |
| Clonality | **Polyclonal** |
| Isotype | **Rabbit IgG** |
| Calculated MW | **41493** |
| Antigen Region | **333-362** |

### CMKOR1 Antibody (C-term) - Additional Information

**Gene ID** 57007

**Other Names**
Atypical chemokine receptor 3, C-X-C chemokine receptor type 7, CXC-R7, CXCR-7, Chemokine orphan receptor 1, G-protein coupled receptor 159, G-protein coupled receptor RDC1 homolog, RDC-1, ACKR3, CMKOR1, CXCR7, GPR159, RDC1

**Target/Specificity**
This CMKOR1 antibody is generated from rabbits immunized with a KLH conjugated synthetic peptide between 333-362 amino acids from the C-terminal region of human CMKOR1.

**Dilution**
FC~~1:10~50
WB~~1:1000
E~~Use at an assay dependent concentration.

**Format**
Purified polyclonal antibody supplied in PBS with 0.09% (W/V) sodium azide. This antibody is purified through a protein A column, followed by peptide affinity purification.

**Storage**
Maintain refrigerated at 2-8°C for up to 2 weeks. For long term storage store at -20°C in small aliquots to prevent freeze-thaw cycles.

**Precautions**
CMKOR1 Antibody (C-term) is for research use only and not for use in diagnostic or therapeutic procedures.

### CMKOR1 Antibody (C-term) - Protein Information

**Name** ACKR3 (HGNC:23692)

**Function** Atypical chemokine receptor that controls chemokine levels and localization via high-affinity chemokine binding that is uncoupled from classic ligand-driven signal transduction cascades, resulting instead in chemokine sequestration, degradation, or transcytosis. Also known as interceptor (internalizing receptor) or chemokine-scavenging receptor or chemokine decoy receptor. Acts as a receptor for chemokines CXCL11 and CXCL12/SDF1 (PubMed:16107333, PubMed:19255243, PubMed:19380869, PubMed:20161793, PubMed:22300987). Chemokine binding does not activate G-protein-mediated signal transduction but instead induces beta-arrestin recruitment, leading to ligand internalization and activation of MAPK signaling pathway (PubMed:16940167, PubMed:18653785, PubMed:20018651). Required for regulation of CXCR4 protein levels in migrating interneurons, thereby adapting their chemokine responsiveness (PubMed:16940167, PubMed:18653785). In glioma cells, transduces signals via MEK/ERK pathway, mediating resistance to apoptosis. Promotes cell growth and survival (PubMed:16940167, PubMed:20388803). Not involved in cell migration, adhesion or proliferation of normal hematopoietic progenitors but activated by CXCL11 in malignant hemapoietic cells, leading to phosphorylation of ERK1/2 (MAPK3/MAPK1) and enhanced cell adhesion and migration (PubMed:17804806, PubMed:18653785, PubMed:19641136, PubMed:20887389). Plays a regulatory role in CXCR4-mediated activation of cell surface integrins by CXCL12 (PubMed:18653785). Required for heart valve development (PubMed:17804806). Regulates axon guidance in the oculomotor system through the regulation of CXCL12 levels (PubMed:31211835). Acts as a receptor for SHLP2, mediating its effects on activation of proopiomelanocortin neurons in the arcuate nucleus of the hypothalamus which leads to suppression of food intake and increased energy expenditure (PubMed:37468558).

**Cellular Location**
Cell membrane; Multi-pass membrane protein. Early endosome. Recycling endosome. Note=Predominantly localizes to endocytic vesicles, and upon stimulation by the ligand is internalized via clathrin-coated pits in a beta-arrestin-dependent manner. Once internalized, the ligand dissociates from the receptor, and is targeted to degradation while the receptor is recycled back to the cell membrane.

**Tissue Location**
Expressed in monocytes, basophils, B-cells, umbilical vein endothelial cells (HUVEC) and B-lymphoblastoid cells Lower expression detected in CD4+ T-lymphocytes and natural killer cells. In the brain, detected in endothelial cells and capillaries, and in mature neurons of the frontal cortex and hippocampus. Expressed in tubular formation in the kidney. Highly expressed in astroglial tumor endothelial, microglial and glioma cells. Expressed at low levels in normal CD34+ progenitor cells, but at very high levels in several myeloid malignant cell lines. Expressed in breast carcinomas but not in normal breast tissue (at protein level).

## CMKOR1 Antibody (C-term) - Protocols

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

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

## CMKOR1 Antibody (C-term) - Images

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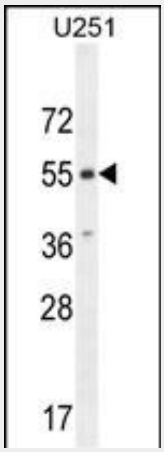
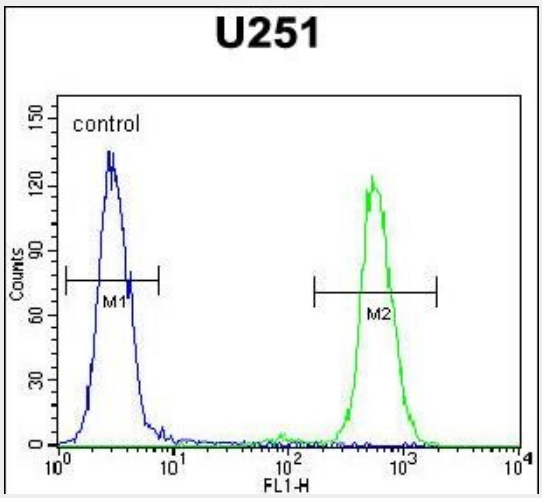



CMKOR1 Antibody (C-term) (Cat. #AP10486b) western blot analysis in U251 cell line lysates (35ug/lane).This demonstrates the CMKOR1 antibody detected the CMKOR1 protein (arrow).




CMKOR1 Antibody (C-term) (Cat. #AP10486b) flow cytometric analysis of U251 cells (right histogram) compared to a negative control cell (left histogram).FITC-conjugated goat-anti-rabbit secondary antibodies were used for the analysis.

**CMKOR1 Antibody (C-term) - Background**

CXCR7 encodes a member of the G-protein coupled receptor family. Although this protein was earlier thought to be a receptor for vasoactive intestinal peptide (VIP), it is now considered to be an orphan receptor, in that its endogenous ligand has not been identified. The protein is also a coreceptor for human immunodeficiency viruses (HIV). Translocations involving this gene and HMGA2 on chromosome 12 have been observed in lipomas. [provided by RefSeq].

**CMKOR1 Antibody (C-term) - References**

Berahovich, R.D., et al. J. Immunol. 185(9):5130-5139(2010)
Watanabe, K., et al. Arthritis Rheum. 62(11):3211-3220(2010)
Hattermann, K., et al. Cancer Res. 70(8):3299-3308(2010)
Miekus, K., et al. Folia Histochem. Cytobiol. 48(1):104-111(2010)
Zheng, K., et al. J. Exp. Clin. Cancer Res. 29, 31 (2010) :