

**CEACAM Antibody (N-term) Blocking peptide**  
Synthetic peptide  
Catalog # BP7364a**Specification**

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**CEACAM Antibody (N-term) Blocking peptide - Product Information**Primary Accession [P13688](#)**CEACAM Antibody (N-term) Blocking peptide - Additional Information**

Gene ID 634

**Other Names**

Carcinoembryonic antigen-related cell adhesion molecule 1, Biliary glycoprotein 1, BGP-1, CD66a, CEACAM1, BGP, BGP1

**Target/Specificity**

The synthetic peptide sequence used to generate the antibody [AP7364a](/products/AP7364a) was selected from the N-term region of human CEACAM. 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.

**CEACAM Antibody (N-term) Blocking peptide - Protein Information**Name CEACAM1 ([HGNC:1814](#))**Function**

[Isoform 1]: Cell adhesion protein that mediates homophilic cell adhesion in a calcium-independent manner (By similarity). Plays a role as coinhibitory receptor in immune response, insulin action and functions also as an activator during angiogenesis (PubMed:[18424730](http://www.uniprot.org/citations/18424730), PubMed:[23696226](http://www.uniprot.org/citations/23696226), PubMed:[25363763](http://www.uniprot.org/citations/25363763)). Its coinhibitory receptor function is phosphorylation- and PTPN6 -dependent, which in turn, suppress signal transduction of associated receptors by dephosphorylation of their downstream effectors. Plays a role in immune response, of T cells, natural killer (NK) and neutrophils (PubMed:[18424730](http://www.uniprot.org/citations/18424730), PubMed:[23696226](http://www.uniprot.org/citations/23696226)). Upon

TCR/CD3 complex stimulation, inhibits TCR-mediated cytotoxicity by blocking granule exocytosis by mediating homophilic binding to adjacent cells, allowing interaction with and phosphorylation by LCK and interaction with the TCR/CD3 complex which recruits PTPN6 resulting in dephosphorylation of CD247 and ZAP70 (PubMed:<a href="http://www.uniprot.org/citations/18424730" target="\_blank">18424730</a>). Also inhibits T cell proliferation and cytokine production through inhibition of JNK cascade and plays a crucial role in regulating autoimmunity and anti-tumor immunity by inhibiting T cell through its interaction with HAVCR2 (PubMed:<a href="http://www.uniprot.org/citations/25363763" target="\_blank">25363763</a>). Upon natural killer (NK) cells activation, inhibit KLRK1-mediated cytotoxicity of CEACAM1-bearing tumor cells by trans-homophilic interactions with CEACAM1 on the target cell and lead to cis-interaction between CEACAM1 and KLRK1, allowing PTPN6 recruitment and then VAV1 dephosphorylation (PubMed:<a href="http://www.uniprot.org/citations/23696226" target="\_blank">23696226</a>). Upon neutrophils activation negatively regulates IL1B production by recruiting PTPN6 to a SYK-TLR4-CEACAM1 complex, that dephosphorylates SYK, reducing the production of reactive oxygen species (ROS) and lysosome disruption, which in turn, reduces the activity of the inflammasome. Down-regulates neutrophil production by acting as a coinhibitory receptor for CSF3R by down-regulating the CSF3R-STAT3 pathway through recruitment of PTPN6 that dephosphorylates CSF3R (By similarity). Also regulates insulin action by promoting INS clearance and regulating lipogenesis in liver through regulating insulin signaling (By similarity). Upon INS stimulation, undergoes phosphorylation by INSR leading to INS clearance by increasing receptor-mediated insulin endocytosis. This internalization promotes interaction with FASN leading to receptor-mediated insulin degradation and to reduction of FASN activity leading to negative regulation of fatty acid synthesis. INSR-mediated phosphorylation also provokes a down-regulation of cell proliferation through SHC1 interaction resulting in decrease coupling of SHC1 to the MAPK3/ERK1-MAPK1/ERK2 and phosphatidylinositol 3-kinase pathways (By similarity). Functions as activator in angiogenesis by promoting blood vessel remodeling through endothelial cell differentiation and migration and in arteriogenesis by increasing the number of collateral arteries and collateral vessel calibers after ischemia. Also regulates vascular permeability through the VEGFR2 signaling pathway resulting in control of nitric oxide production (By similarity). Down-regulates cell growth in response to EGF through its interaction with SHC1 that mediates interaction with EGFR resulting in decrease coupling of SHC1 to the MAPK3/ERK1-MAPK1/ERK2 pathway (By similarity). Negatively regulates platelet aggregation by decreasing platelet adhesion on type I collagen through the GPVI-FcRgamma complex (By similarity). Inhibits cell migration and cell scattering through interaction with FLNA; interferes with the interaction of FLNA with RALA (PubMed:<a href="http://www.uniprot.org/citations/16291724" target="\_blank">16291724</a>). Mediates bile acid transport activity in a phosphorylation dependent manner (By similarity). Negatively regulates osteoclastogenesis (By similarity).

### Cellular Location

[Isoform 1]: Cell membrane {ECO:0000250|UniProtKB:P16573}; Single-pass type I membrane protein {ECO:0000250|UniProtKB:P16573}. Lateral cell membrane {ECO:0000250|UniProtKB:P16573}. Apical cell membrane {ECO:0000250|UniProtKB:P16573}. Basal cell membrane {ECO:0000250|UniProtKB:P16573}. Cell junction. Cell junction, adherens junction {ECO:0000250|UniProtKB:P16573}. Note=Canalicular domain of hepatocyte plasma membranes. Found as a mixture of monomer, dimer and oligomer in the plasma membrane. Occurs predominantly as cis-dimers and/or small cis-oligomers in the cell junction regions. Found as dimer in the solution. Predominantly localized to the lateral cell membranes {ECO:0000250|UniProtKB:P16573} [Isoform 3]: Secreted [Isoform 5]: Cell membrane; Single-pass type I membrane protein [Isoform 7]: Cell membrane; Single-pass type I membrane protein Cell projection, microvillus membrane {ECO:0000250|UniProtKB:P31809}; Single-pass type I membrane protein. Apical cell membrane; Single-pass type I membrane protein. Note=Localized to the apical glycocalyx surface (PubMed:10436421). Colocalizes with CEACAM20 at the apical brush border of intestinal cells {ECO:0000250|UniProtKB:P31809, ECO:0000269|PubMed:10436421}

### Tissue Location

Expressed in columnar epithelial cells of the colon (at protein level) (PubMed:10436421). The

predominant forms expressed by T cells are those containing a long cytoplasmic domain (PubMed:18424730). Expressed in granulocytes and lymphocytes Leukocytes only express isoforms 6 and isoform 1 (PubMed:11994468)

### **CEACAM Antibody (N-term) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

### **CEACAM Antibody (N-term) Blocking peptide - Images**

### **CEACAM Antibody (N-term) Blocking peptide - Background**

CEACAM is a member of the carcinoembryonic antigen (CEA) family, which belongs to the immunoglobulin superfamily. The protein was originally described in bile ducts of liver as biliary glycoprotein. Subsequently, it was found to be a cell-cell adhesion molecule detected on leukocytes, epithelia, and endothelia. The protein mediates cell adhesion via homophilic as well as heterophilic binding to other proteins of the subgroup. Multiple cellular activities have been attributed to the protein, including roles in the differentiation and arrangement of tissue three-dimensional structure, angiogenesis, apoptosis, tumor suppression, metastasis, and the modulation of innate and adaptive immune responses.

### **CEACAM Antibody (N-term) Blocking peptide - References**

Gambichler,T., Am. J. Clin. Pathol. 131 (6), 782-787 (2009)Li,C., Exp. Cell Res. 315 (7), 1225-1233 (2009)Nouvion,A.L., Med Sci (Paris) 25 (3), 247-252 (2009)