

BAI1 Antibody (C-term) Blocking Peptide

Synthetic peptide Catalog # BP8170a

Specification

BAI1 Antibody (C-term) Blocking Peptide - Product Information

Primary Accession O14514
Other Accession BAIL HUMAN

BAI1 Antibody (C-term) Blocking Peptide - Additional Information

Gene ID 575

Other Names

Brain-specific angiogenesis inhibitor 1, BAI1

Target/Specificity

The synthetic peptide sequence used to generate the antibody AP8170a was selected from the C-term region of human BAI1 . 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.

BAI1 Antibody (C-term) Blocking Peptide - Protein Information

Name ADGRB1 (HGNC:943)

Function

Phosphatidylserine receptor which enhances the engulfment of apoptotic cells (PubMed:24509909). Also mediates the binding and engulfment of Gram-negative bacteria (PubMed:26838550). Stimulates production of reactive oxygen species by macrophages in response to Gram-negative bacteria, resulting in enhanced microbicidal macrophage activity (PubMed:26838550). In the gastric mucosa, required for recognition and engulfment of apoptotic gastric epithelial cells (PubMed:24509909). Promotes myoblast fusion (By similarity). Activates the Rho pathway in a G-protein-dependent manner (PubMed:23782696).



Inhibits MDM2-mediated ubiquitination and degradation of DLG4/PSD95, promoting DLG4 stability and regulating synaptic plasticity (By similarity). Required for the formation of dendritic spines by ensuring the correct localization of PARD3 and TIAM1 (By similarity). Potent inhibitor of angiogenesis in brain and may play a significant role as a mediator of the p53/TP53 signal in suppression of glioblastoma (PubMed:11875720).

Cellular Location

Cell membrane; Multi-pass membrane protein. Cell projection, phagocytic cup {ECO:0000250|UniProtKB:Q3UHD1}. Cell junction, focal adhesion {ECO:0000250|UniProtKB:Q3UHD1}. Cell projection, dendritic spine {ECO:0000250|UniProtKB:C0HL12}. Postsynaptic density {ECO:0000250|UniProtKB:Q3UHD1} [Vasculostatin-40]: Secreted

Tissue Location

Expressed in brain (at protein level) (PubMed:12074842, PubMed:12507886). Expressed on mononuclear phagocytes and monocyte-derived macrophages in the gastric mucosa (at protein level) (PubMed:24509909). Expressed in normal pancreatic tissue but not in pancreatic tumor tissue (PubMed:11875720). Reduced or no expression is observed in some glioblastomas (PubMed:12507886)

BAI1 Antibody (C-term) Blocking Peptide - Protocols

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

• Blocking Peptides

BAI1 Antibody (C-term) Blocking Peptide - Images

BAI1 Antibody (C-term) Blocking Peptide - Background

Angiogenesis is controlled by a local balance between stimulators and inhibitors of new vessel growth and is suppressed under normal physiologic conditions. Angiogenesis has been shown to be essential for growth and metastasis of solid tumors. In order to obtain blood supply for their growth, tumor cells are potently angiogenic and attract new vessels as results of increased secretion of inducers and decreased production of endogenous negative regulators. BAI1 contains at least one 'functional' p53-binding site within an intron, and its expression has been shown to be induced by wildtype p53. There are two other brain-specific angiogenesis inhibitor genes, designated BAI2 and BAI3 which along with BAI1 have similar tissue specificities and structures, however only BAI1 is transcriptionally regulated by p53. BAI1 is postulated to be a member of the secretin receptor family, an inhibitor of angiogenesis and a growth suppressor of glioblastomas.

BAI1 Antibody (C-term) Blocking Peptide - References

Kaur, B., et al., Am. J. Pathol. 162(1):19-27 (2003).Mori, K., et al., Neurosci. Res. 43(1):69-74 (2002).Duda, D.G., et al., Br. J. Cancer 86(3):490-496 (2002).Shiratsuchi, T., et al., Biochem. Biophys. Res. Commun. 247(3):597-604 (1998).Fukushima, Y., et al., Int. J. Oncol. 13(5):967-970 (1998).