

小鼠抗绒毛蛋白单克隆抗体

产品货号： mlR0137

英文名称： Villin

中文名称： 小鼠抗绒毛蛋白单克隆抗体

别名： VIL; VIL1; Villin 1; Villin-1; Villin1; D2S1471; OTTHUMP00000164145; VILI_HUMAN.

研究领域： 肿瘤 免疫学 神经生物学 内分泌病

抗体来源： Mouse

克隆类型： Monoclonal

克隆号： 5F9

交叉反应： Human,

产品应用： IHC-P=1:100-500 IHC-F=1:100-500 ICC=1:100-500 IF=1:100-500 (石蜡切片需做抗原修复)

not yet tested in other applications.

optimal dilutions/concentrations should be determined by the end user.

分子量： 93kDa

细胞定位： 细胞浆

性状： Lyophilized or Liquid

浓度： 1mg/ml

免疫原： KLH conjugated synthetic peptide derived from human Villin :

亚型： IgG2b

纯化方法 : affinity purified by Protein A

储存液 : 0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.

保存条件 : Store at -20 ° C for one year. Avoid repeated freeze/thaw cycles. The lyophilized antibody is stable at room temperature for at least one month and for greater than a year when kept at -20° C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 ° C.

PubMed : PubMed

产品介绍 : Villin can cap, nucleate, sever and bundle actin in a calcium and phosphoinositide regulated manner. It is associated with the microvillar actin core bundle of intestinal and renal brush border implicated in adsorption. Villin is composed of six repeats, each containing 150 residues that together constitute the core domain followed by the carboxyl terminal headpiece domain of 87 residues. The core domain retains the calcium dependent capping nucleating and severing activity, whereas the headpiece domain contributes towards actin filament bundling and binding F actin, independently of Calcium.

Function:

Epithelial cell-specific Ca(2+)-regulated actin-modifying protein that modulates the reorganization of microvillar actin filaments. Plays a role in the actin nucleation, actin filament bundle assembly, actin filament capping and severing. Binds phosphatidylinositol 4,5-bisphosphate (PIP2) and lysophosphatidic acid (LPA); binds LPA with higher affinity than PIP2. Binding to LPA increases its phosphorylation by SRC and inhibits all actin-modifying activities. Binding to PIP2 inhibits actin-capping and -severing activities but enhances actin-bundling activity. Regulates the intestinal epithelial cell morphology, cell invasion, cell migration and apoptosis. Protects against apoptosis induced by dextran sodium sulfate (DSS) in the gastrointestinal epithelium. Appears to regulate cell death by maintaining mitochondrial integrity. Enhances hepatocyte growth factor (HGF)-induced epithelial cell motility, chemotaxis and wound repair. Upon *S.flexneri* cell infection, its actin-severing activity enhances actin-based motility of the bacteria and plays a role during the dissemination.

Subunit:

Monomer. Homodimer; homodimerization is necessary for actin-bundling. Associates with F-actin; phosphorylation at tyrosines residues decreases the association with F-actin. Interacts (phosphorylated at C-

terminus tyrosine phosphorylation sites) with PLCG1 (via the SH2 domains). Interacts (phosphorylated form) with PLCG1; the interaction is enhanced by hepatocyte growth factor (HGF) (By similarity).

Subcellular Location:

Cytoplasm, cytoskeleton. Cell projection, lamellipodium. Cell projection, ruffle. Cell projection, microvillus. Cell projection, filopodium tip (By similarity). Cell projection, filopodium (By similarity). Note=Relocalized in the tip of cellular protrusions and filopodial extensions upon infection with *S.flexneri* in primary intestinal epithelial cells (IEC) and in the tail-like structures forming the actin comets of *S.flexneri*. Redistributed to the leading edge of hepatocyte growth factor (HGF)-induced lamellipodia (By similarity). Rapidly redistributed to ruffles and lamellipodia structures in response to autotaxin, lysophosphatidic acid (LPA) and epidermal growth factor (EGF) treatment.

Tissue Specificity:

Specifically expressed in epithelial cells. Major component of microvilli of intestinal epithelial cells and kidney proximal tubule cells. Expressed in canalicular microvilli of hepatocytes (at protein level).

Post-translational modifications:

Tyrosine phosphorylation is induced by epidermal growth factor (EGF) and stimulates cell migration (By similarity). Phosphorylated on tyrosine residues by SRC. The unphosphorylated form increases the initial rate of actin-nucleating activity, whereas the tyrosine-phosphorylated form inhibits actin-nucleating activity, enhances actin-bundling activity and enhances actin-severing activity by reducing high Ca(2+) requirements. The tyrosine-phosphorylated form does not regulate actin-capping activity. Tyrosine phosphorylation is essential for cell migration: tyrosine phosphorylation sites in the N-terminus half regulate actin reorganization and cell morphology, whereas tyrosine phosphorylation sites in the C-terminus half regulate cell migration via interaction with PLCG1.

DISEASE:

Note=Biliary atresia is a chronic and progressive cholestatic liver disease of childhood characterized by an abnormal villin gene expression and severe malformation of canalicular microvillus structure.

Similarity:

Belongs to the villin/gelsolin family.

Contains 6 gelsolin-like repeats.

Contains 1 HP (headpiece) domain

SWISS:

P09327

Gene ID:

7429

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

产品图片

