

Product datasheet for AP26021PU-N

KRIT1 Rabbit Polyclonal Antibody

Product data:

OriGene Technologies, Inc.

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| Product Type: | Primary Antibodies |
|------------------------|---|
| Applications: | IF, WB |
| Recommended Dilution: | Western Blot: 1-5 μg/ml. Immunofluorescence: 1-10 μg/ml. Immunohistochemistry: 1/200. |
| Reactivity: | Human |
| Host: | Rabbit |
| lsotype: | IgG |
| Clonality: | Polyclonal |
| Immunogen: | Highly pure (>95%) recombinant Human CCM-1 (Met1-Ser736) derived from <i>E. coli</i> fused to a C-teminal His-tag (6 x His) (<i>CatNo</i> AR26002PU-N). |
| Specificity: | This antibody is anti-His depleated. It detects KRIT1 / CCM1. |
| Formulation: | 5mM PBS, pH 7.2 State: Purified State: Lyophilized purified IgG fraction |
| Reconstitution Method: | Restore in sterile water to a concentration of 0.1-1.0 mg/ml. Centrifuge vial prior to opening. |
| Purification: | Protein A Chromatography |
| Conjugation: | Unconjugated |
| Storage: | Prior to reconstitution store at 2-8°C. Following reconstitution store undiluted at 2-8°C for one month or (in aliquots) at -20°C for longer. Avoid repeated freezing and thawing. |
| Stability: | Shelf life: one year from despatch. |
| Gene Name: | KRIT1, ankyrin repeat containing |
| Database Link: | Entrez Gene 889 Human O00522 |



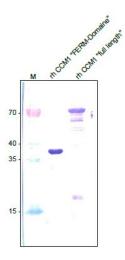
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GRIGENE KRIT1 Rabbit Polyclonal Antibody – AP26021PU-N

Background: Cerebral Cavernous Malformations (CCM) are frequent vascular abnormalities caused by mutations in one of the CCM genes. CCM-1 (also known as KRIT1) stabilizes endothelial junctions and is essential for vascular morphogenesis in mouse embryos. However, cellular functions of CCM-1 during the early steps of the CCM pathogenesis remain unknown. It was shown that CCM-1 represents an antiangiogenic protein to keep the human endothelium quiescent. CCM-1 inhibits endothelial proliferation, apoptosis, migration, lumen formation, and sprouting angiogenesis in primary human endothelial cells. CCM-1 strongly induces DLL4-NOTCH signaling, which promotes AKT phosphorylation but reduces phosphorylation of the mitogen-activated protein kinase ERK. Consistently, blocking of NOTCH activity alleviates CCM-1 effects. ERK phosphorylation is increased in human CCM lesions. Transplantation of CCM-1-silenced human endothelial cells into SCID mice recapitulates hallmarks of the CCM pathology and serves as a unique CCM model system.

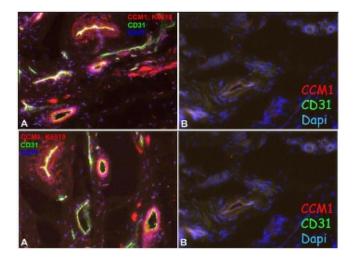
Synonyms:Krev interaction trapped 1Protein Families:Druggable Genome

Product images:



Western analysis of recombinant Human CCM-1 (FERM domain) and recombinant Human full length CCM-1 using a Rabbit polyclonal anti-Human CCM-1 antibody generated against the recombinant FERM domain of Human CCM-1.

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Immunofluorescence staining of Human foreskin (Cryo-section of unfixed tissue) with anti-CCM1 Antibody (red, dilution: 1/50). Costaining of endothelial cells with anti-CD31 (green). Note specific staining in the wall of a subset of vessel. Nuclei counter-stained with Dapi (blue). Specimen provided by Prof. Dr. J. Wilting and Dr. K. Buttler, Goettingen.

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