

## Product datasheet for AP26021PU-L

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# **KRIT1 Rabbit Polyclonal Antibody**

**Product data:** 

**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
Isotype: IgG

Clonality: Polyclonal

Immunogen: Highly pure (>95%) recombinant Human CCM-1 (Met1-Ser736) derived from *E. coli* fused to a

C-teminal His-tag (6 x His) (Cat.-No 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

000522





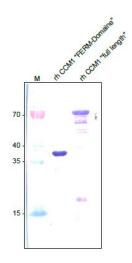
Background:

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 1

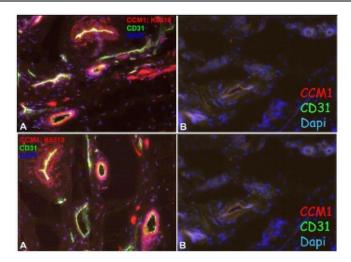
**Protein Families:** Druggable Genome

### **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.





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.