

Recombinant Human KRIT1 protein ab113196

1 Image

Description

Product name	Recombinant Human KRIT1 protein
Purity	> 90 % SDS-PAGE. Purity is >90%, by SDS-PAGE and silver stain.
Expression system	Escherichia coli
Accession	<u>O00522-2</u>
Protein length	Protein fragment
Animal free	No
Nature	Recombinant
Species	Human
Sequence	MLLKEAINKPYEKVRIYRMDGSYRSVELKHGNNTTVQQIME GMRLSQETQ QYFTWICSENLSLQLKPYHKPLQHVRDWPEILAEELTNLDP QRETPQLFL RRDVRLPLEVEKQIEDPLAILILFDEARYNLLKGFYTAPDAK LITLASLL LQVYGNYESKKHKQGFLNEENLKSIPVTKLKSAPHWT NRILHEYKNL STSEGVSKEMHHLQRMFLQNCWEIPTYGAAFFTGGQIFTKA SPSNHKVIPV YVGVNIKGLHLLNMETKALLISLKYGCFMWQLGDDTCTCFQI HSMENKMSF IVHTKQAGLVVKLLMKLNGQLMPTERNLSLE
Predicted molecular weight	39 kDa including tags
Amino acids	203 to 529
Tags	His tag C-Terminus

Specifications

Our **Abpromise guarantee** covers the use of **ab113196** in the following tested applications.

The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Applications	SDS-PAGE
Form	Lyophilized

Preparation and Storage

Stability and Storage

Shipped at 4°C. Upon delivery aliquot. Store at -20°C. Avoid freeze / thaw cycle.

Constituent: 0.18% Sodium chloride

Reconstitution

ab113196 is soluble in water and most aqueous buffers and should be reconstituted in PBS or medium containing at least 0.1% Human or BSA to a concentration not lower than 50 µg/ml.

General Info

Function

Negative regulator of angiogenesis. Inhibits endothelial proliferation, apoptosis, migration, lumen formation and sprouting angiogenesis in primary endothelial cells. Promotes AKT phosphorylation in a NOTCH-dependent and independent manner, and inhibits EKR1/2 phosphorylation indirectly through activation of the DELTA-NOTCH cascade. Acts in concert with CDH5 to establish and maintain correct endothelial cell polarity and vascular lumen and these effects are mediated by recruitment and activation of the Par polarity complex and RAP1B. Required for the localization of phosphorylated PRKCZ, PARD3, TIAM1 and RAP1B to the cell junction. Plays an important role in the maintenance of the intracellular reactive oxygen species (ROS) homeostasis to prevent oxidative cellular damage. Regulates the homeostasis of intracellular ROS through an antioxidant pathway involving FOXO1 and SOD2. Facilitates the down-regulation of cyclin D1 levels required for cell transition from proliferative growth to quiescence by preventing the accumulation of intracellular ROS through the modulation of FOXO1 and SOD2 levels.

Tissue specificity

Low levels in brain. Very weak expression found in heart and muscle.

Involvement in disease

Defects in KRIT1 are the cause of cerebral cavernous malformations type 1 (CCM1) [MIM:116860]. Cerebral cavernous malformations (CCMs) are congenital vascular anomalies of the central nervous system that can result in hemorrhagic stroke, seizures, recurrent headaches, and focal neurologic deficits. CCMs have an incidence of 0.1%-0.5% in the general population and usually present clinically during the 3rd to 5th decade of life. The lesions are characterized by grossly enlarged blood vessels consisting of a single layer of endothelium and without any intervening neural tissue, ranging in diameter from a few millimeters to several centimeters.

Sequence similarities

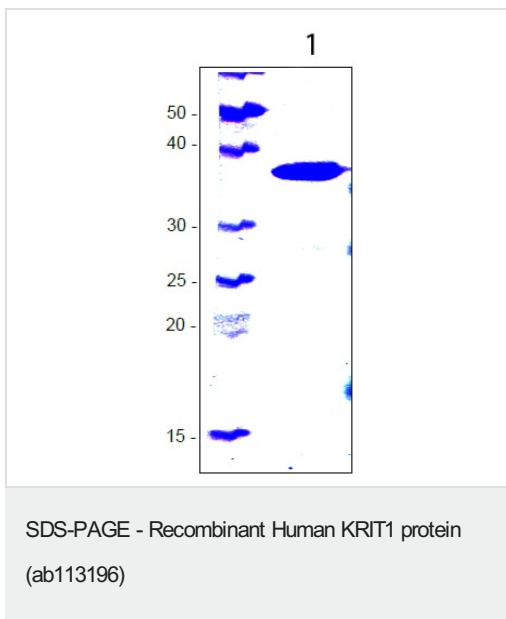
Contains 4 ANK repeats.

Contains 1 FERM domain.

Cellular localization

Membrane. Cell junction. KRIT1 and CDH5 reciprocally regulate their localization to endothelial cell-cell junctions.

Images



SDS-PAGE analysis of recombinant human KRIT1 protein fragment (ab113196). Sample was loaded in 15% SDS-polyarylamide gel under reducing conditions and stained with Coomassie blue.

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