abcam

Product datasheet

Recombinant Human Vinculin protein (Tagged) ab238349

1 Image

Description

Product name Recombinant Human Vinculin protein (Tagged)

Purity > 90 % SDS-PAGE.

Expression system Escherichia coli

Accession P18206-1

Protein length Protein fragment

Animal free No

Nature Recombinant

Species Human

Sequence PVFHTRTIESILEPVAQQISHLVIMHEEGEVDGKAIPDLTAP

VAAVQAAV

SNLVRVGKETVQTTEDQILKRDMPPAFIKVENACTKLVQA

AQMLQSDPYS

VPARDYLIDGSRGILSGTSDLLLTFDEAEVRKIIRVCKGILEY

LTVAEVV

ETMEDLVTYTKNLGPGMTKMAKMIDERQQELTHQEHRVM

LVNSMNTVKEL

LPVLISAMKIFVTTKNSKNQGIEEALKNRNFTVE

Predicted molecular weight 53 kDa including tags

Amino acids 2 to 235

Tags GST tag N-Terminus

Specifications

Our <u>Abpromise guarantee</u> covers the use of ab238349 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 Liquid

Preparation and Storage

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Stability and Storage

Shipped at 4°C. Store at -20°C or -80°C. Avoid freeze / thaw cycle.

pH: 7.2

Constituents: Tris buffer, 50% Glycerol (glycerin, glycerine)

General Info

Function

Actin filament (F-actin)-binding protein involved in cell-matrix adhesion and cell-cell adhesion. Regulates cell-surface E-cadherin expression and potentiates mechanosensing by the E-cadherin complex. May also play important roles in cell morphology and locomotion.

Tissue specificity

Involvement in disease

Metavinculin is muscle-specific.

Defects in VCL are the cause of cardiomyopathy dilated type 1W (CMD1W) [MIM:611407]. Dilated cardiomyopathy is a disorder characterized by ventricular dilation and impaired systolic function, resulting in congestive heart failure and arrhythmia. Patients are at risk of premature death.

Defects in VCL are the cause of cardiomyopathy familial hypertrophic type 15 (CMH15) [MIM:613255]. It is a hereditary heart disorder characterized by ventricular hypertrophy, which is usually asymmetric and often involves the interventricular septum. The symptoms include dyspnea, syncope, collapse, palpitations, and chest pain. They can be readily provoked by exercise. The disorder has inter- and intrafamilial variability ranging from benign to malignant forms with high risk of cardiac failure and sudden cardiac death.

Sequence similarities

Domain

Belongs to the vinculin/alpha-catenin family.

Exists in at least two conformations. When in the closed, 'inactive' conformation, extensive interactions between the head and tail domains prevent detectable binding to most of its ligands. It takes on an 'active' conformation after cooperative and simultaneous binding of two different ligands. This activation involves displacement of the head-tail interactions and leads to a significant accumulation of ternary complexes. The active form then binds a number of proteins that have both signaling and structural roles that are essential for cell adhesion.

The N-terminal globular head (Vh) comprises of subdomains D1-D4. The C-terminal tail (Vt) binds F-actin and cross-links actin filaments into bundles. An intramolecular interaction between Vh and Vt masks the F-actin-binding domain located in Vt. The binding of talin and alpha-actinin to the D1 subdomain of vinculin induces a helical bundle conversion of this subdomain, leading to the disruption of the intramolecular interaction and the exposure of the cryptic F-actin-binding domain of Vt. Vt inhibits actin filament barbed end elongation without affecting the critical concentration of actin assembly.

Post-translational modifications

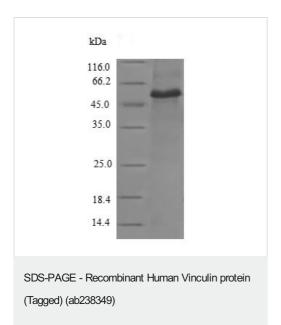
Phosphorylated; on serines, threonines and tyrosines. Phosphorylation on Tyr-1133 in activated platelets affects head-tail interactions and cell spreading but has no effect on actin binding nor on localization to focal adhesion plaques.

Aceylated; mainly by myristic acid but also small amount of palmitic acid.

Cellular localization

Cytoplasm > cytoskeleton. Cell junction > adherens junction. Cell membrane. Cytoplasmic face of adhesion plaques. Recruitment to cell-cell junctions occurs in a myosin ll-dependent manner. Interaction with CTNNB1 is necessary for its localization to the cell-cell junctions.

Images



ab238349 analyzed by (Tris-Glycine gel) discontinuous SDS-PAGE (reduced) with 5% enrichment gel and 15% separation gel.

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