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Product datasheet

Anti-Vinculin antibody - C-terminal ab227783

1 Image

Overview

Product name	Anti-Vinculin antibody - C-terminal
Description	Rabbit polyclonal to Vinculin - C-terminal
Host species	Rabbit
Tested applications	**Suitable for:** IHC-P
Species reactivity	**Reacts with:** Human **Predicted to work with:** Mouse, Rat, Rabbit, Chicken, Cow, Dog, Pig
Immunogen	Synthetic peptide within Human Vinculin aa 850-950 (C terminal). The exact sequence is proprietary. Database link: P18206
Positive control	IHC-P: Human pancreas tissue.
General notes	**This product is FOR RESEARCH USE ONLY. For commercial use, please contact partnerships@abcam.com.**

Properties

Form	Liquid
Storage instructions	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Upon delivery aliquot. Store at -20°C long term. Avoid freeze / thaw cycle.
Storage buffer	pH: 7.6 Preservative: 0.1% Sodium azide Constituents: PBS, 1% BSA
Purity	Immunogen affinity purified
Clonality	Polyclonal
Isotype	IgG

Applications

Our Abpromise guarantee covers the use of **ab227783** in the following tested applications.

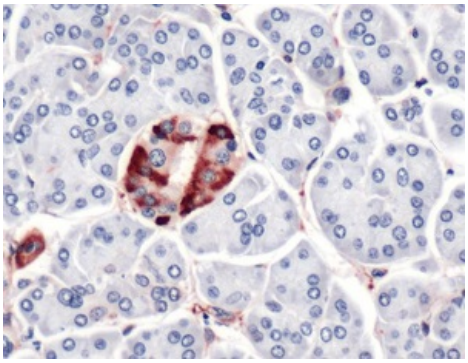
The application notes include recommended starting dilutions; optimal dilutions/concentrations should be determined by the end user.

Application	Abreviews	Notes
IHC-P		1/200. Perform heat mediated antigen retrieval with citrate buffer pH 6 before commencing with IHC staining protocol.

Target

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	Metavinculin is muscle-specific.
Involvement in disease	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	Belongs to the vinculin/alpha-catenin family.
Domain	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 II-dependent manner. Interaction with CTNNB1 is necessary for its localization to the cell-cell junctions.

Images



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Vinculin antibody - C-terminal (ab227783)

Formalin-fixed, paraffin-embedded human pancreas tissue stained for Vinculin with ab227783 (1/200 dilution) in immunohistochemical analysis.

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