




Product datasheet

Anti-Caveolin-3 antibody - N-terminal ab189349

2 Images

Overview

Product name	Anti-Caveolin-3 antibody - N-terminal
Description	Goat polyclonal to Caveolin-3 - N-terminal
Host species	Goat
Tested applications	Suitable for: WB, IHC-P
Species reactivity	Reacts with: Human Predicted to work with: Rabbit, Horse, Cow, Dog, Pig, Monkey, Gorilla 
Immunogen	Synthetic peptide corresponding to Human Caveolin-3 aa 1-100 (N terminal) (Cysteine residue). (NP_203123.1) Database link: P56539 <div>  Run BLAST with  Run BLAST with </div>
Positive control	Human, Mouse and Rat Heart lysates: Human skeletal muscle tissue.
General notes	<p>The Life Science industry has been in the grips of a reproducibility crisis for a number of years. Abcam is leading the way in addressing this with our range of recombinant monoclonal antibodies and knockout edited cell lines for gold-standard validation. Please check that this product meets your needs before purchasing.</p> <p>If you have any questions, special requirements or concerns, please send us an inquiry and/or contact our Support team ahead of purchase. Recommended alternatives for this product can be found below, along with publications, customer reviews and Q&As</p>

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: 7.30 Preservative: 0.02% Sodium azide Constituents: 99% Tris buffered saline, 0.5% BSA
Purity	Immunogen affinity purified
Clonality	Polyclonal
Isotype	IgG

Applications

The Abpromise guarantee

Our **Abpromise guarantee** covers the use of ab189349 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
WB		Use a concentration of 0.3 - 1 µg/ml. Detects a band of approximately 23 kDa (predicted molecular weight: 17 kDa).
IHC-P		Use a concentration of 3.75 µg/ml. Perform heat mediated antigen retrieval before commencing with IHC staining protocol.

Target

Function

May act as a scaffolding protein within caveolar membranes. Interacts directly with G-protein alpha subunits and can functionally regulate their activity. May also regulate voltage-gated potassium channels. Plays a role in the sarcolemma repair mechanism of both skeletal muscle and cardiomyocytes that permits rapid resealing of membranes disrupted by mechanical stress.

Tissue specificity

Expressed predominantly in muscle.

Involvement in disease

Defects in CAV3 are the cause of limb-girdle muscular dystrophy type 1C (LGMD1C) [MIM:607801]. LGMD1C is a myopathy characterized by calf hypertrophy and mild to moderate proximal muscle weakness. LGMD1C inheritance can be autosomal dominant or recessive. Defects in CAV3 are a cause of hyperCKmia (HYPCK) [MIM:123320]. It is a disease characterized by persistent elevated levels of serum creatine kinase without muscle weakness. Defects in CAV3 are a cause of rippling muscle disease (RMD) [MIM:606072]. RMD is a rare disorder characterized by mechanically triggered contractions of skeletal muscle. In RMD, mechanical stimulation leads to electrically silent muscle contractions that spread to neighboring fibers that cause visible ripples to move over the muscle. Defects in CAV3 are a cause of cardiomyopathy familial hypertrophic (CMH) [MIM:192600]; also designated FHC or HCM. Familial hypertrophic cardiomyopathy 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. Defects in CAV3 are the cause of long QT syndrome type 9 (LQT9) [MIM:611818]. Long QT syndromes are heart disorders characterized by a prolonged QT interval on the ECG and polymorphic ventricular arrhythmias. They cause syncope and sudden death in response to exercise or emotional stress. They can present with a sentinel event of sudden cardiac death in infancy. Defects in CAV3 can be a cause of sudden infant death syndrome (SIDS) [MIM:272120]. SIDS is the sudden death of an infant younger than 1 year that remains unexplained after a thorough case investigation, including performance of a complete autopsy, examination of the death scene, and review of clinical history. Pathophysiologic mechanisms for SIDS may include respiratory dysfunction, cardiac dysrhythmias, cardiorespiratory instability, and inborn errors of metabolism, but definitive pathogenic mechanisms precipitating an infant sudden death remain elusive. Long QT syndromes-associated mutations can be responsible for some SIDS cases.

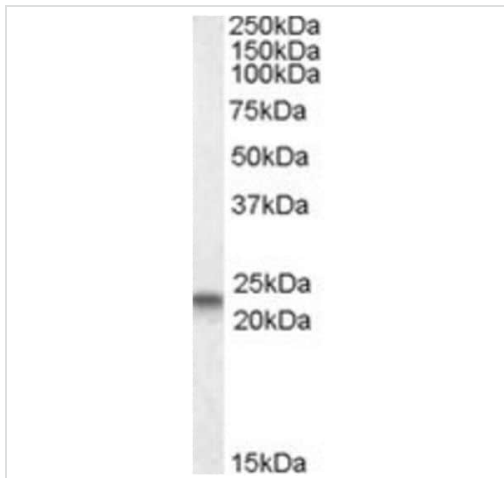
Sequence similarities

Belongs to the caveolin family.

Cellular localization

Golgi apparatus membrane. Cell membrane. Membrane > caveola. Potential hairpin-like structure in the membrane. Membrane protein of caveolae.

Images

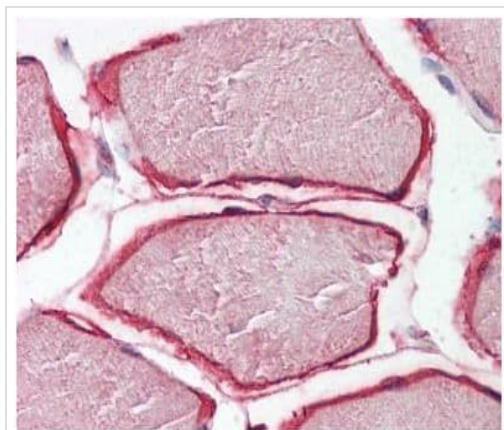


Western blot - Anti-Caveolin-3 antibody - N-terminal (ab189349)

Anti-Caveolin-3 antibody - N-terminal (ab189349) at 0.3 µg/ml + Human Heart lysate in RIPA buffer at 35 µg/ml

Developed using the ECL technique.

Predicted band size: 17 kDa



Immunohistochemistry (Formalin/PFA-fixed paraffin-embedded sections) - Anti-Caveolin-3 antibody - N-terminal (ab189349)

Immunohistochemical analysis of formalin-fixed, paraffin-embedded Human skeletal muscle tissue labeling Caveolin-3 with ab189349 at 3.75 µg/ml.

Please note: All products are "FOR RESEARCH USE ONLY. NOT FOR USE IN DIAGNOSTIC PROCEDURES"

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