

Product datasheet

Mouse Caveolin-3 peptide ab4930

Description

Product name	Mouse Caveolin-3 peptide
Purity	> 70 % HPLC. Peptides are analyzed by Reverse-Phase HPLC (RP-HPLC) in order to determine purity. Identities are confirmed by MALDI-MS.
Accession	<u>P51637</u>
Animal free	No
Nature	Synthetic
Species	Mouse
Sequence	MMTEEHTDLEARIIKDIHC
Amino acids	1 to 19

Specifications

Our **Abpromise guarantee** covers the use of **ab4930** in the following tested applications.

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

Applications	Blocking
Form	Lyophilized
Additional notes	

This peptide may be used for neutralization and control experiments with the polyclonal antibody that reacts with this product and endogenous caveolin-3, catalog **ab2912**. Using a solution of peptide of equal volume and concentration to the corresponding antibody will yield a large molar excess of peptide (~ 70-fold) for competitive inhibition of antibody-protein binding reactions.

Preparation and Storage

Stability and Storage	Shipped at 4°C. Store at +4°C short term (1-2 weeks). Store at -20°C or -80°C. Avoid freeze / thaw cycle.
Reconstitution	>95% pure, lyophilized synthetic peptide. Reconstitute with 0.1 ml of distilled water.

General Info

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	<p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p> <p>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.</p>
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.

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