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Rabbit Anti-phospholamban/FITC Conjugated antibody

SL4197R-FITC

Anti-phospholamban/FITC
FITC标记的受磷蛋白/心脏磷蛋白抗体
Cardiac phospholamban; CMD1P; PLB; PLN; PPLA_HUMAN.
Rabbit
Polyclonal
Human,Mouse,Rat,Pig,Cow,
IF=1:50-200
not yet tested in other applications.
optimal dilutions/concentrations should be determined by the end user.
5.7kDa
The cell membrane <u>Mitochondrion</u>
Lyophilized or Liquid
1mg/ml
KLH conjugated synthetic peptide derived from human phospholamban
IgG
affinity purified by Protein A
0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Store at -20 °C for one year. Avoid repeated freeze/thaw cycles. The lyophilized
antibody is stable at room temperature for at least one month and for greater than a year
when kept at -20°C. When reconstituted in sterile pH 7.4 0.01M PBS or diluent of
antibody the antibody is stable for at least two weeks at 2-4 °C.
background:
The Sarco(endo)plasmic-reticulum (SER) regulatory protein, Phospholamban (PLB), is
a small, plasma membrane-associated phospho-protein found in the SER of cardiac,
smooth and slow-twitch muscle. Believed to assemble into a pentamer, PLB regulates
cardiac contractility and Ca2+ affinity for cardiac SER Ca2+ ATPase (SERCA2a).
Non-phosphorylated PLB associates with SERCA2a, and inhibits Ca2+ reuptake into
the SER. PLB activation occurs when key Serine/Threonine residues in PLB (Ser-10,

Ser-16, Thr-17) are phosphorylated by numerous effectors, which include PKC, PKA, PKG, and CaM kinase. Phosphorylation of PLB causes dissociation from SERCA2a and a subsequent increase in the rate of Ca2+ reuptake into the SER, which accelerates ventricular relaxation.

Function:

Phospholamban has been postulated to regulate the activity of the calcium pump of cardiac sarcoplasmic reticulum.

Subcellular Location:

Mitochondrion membrane; Single-pass membrane protein. Sarcoplasmic reticulum.

Tissue Specificity: Heart.

Post-translational modifications:

Phosphorylated at Thr-17 by CaMK2, and in response to beta-adrenergic stimulation. Phosphorylation by DMPK may stimulate sarcoplasmic reticulum calcium uptake in cardiomyocytes.

DISEASE:

Defects in PLN are the cause of cardiomyopathy dilated type 1P (CMD1P) [MIM:609909]. 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 PLN are the cause of familial hypertrophic cardiomyopathy type 18 (CMH18) [MIM:613874]. CMH18 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.

Similarity:

Belongs to the phospholamban family.

Database links:

Entrez Gene: 5350Human

Entrez Gene: 18821Mouse

Entrez Gene: 64672Rat

Omim: 172405Human

SwissProt: A4IFH6Cow

	SwissProt: P26678Human
	SwissProt: P61014Mouse
	SwissProt: P61013Pig
	SwissProt: P61015Rabbit
	SwissProt: P61016Rat
	Unigene: 170839Human
	Unigene: 34145 Mouse
	<u>Unigene: 9740</u> Rat
	Important Note:
	This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.
	therapeutic or diagnostic applications.
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