



Rabbit Anti-MYBPC3 antibody

SL9868R

Product Name:	MYBPC3
Chinese Name:	心脏肌球蛋白Binding protein抗体
Alias:	C protein cardiac muscle isoform; cardiac muscle isoform; cardiac-type; C-protein; Cardiac MyBP C; Cardiac MyBP-C; Cardiac myosin binding protein C; MYBP C; MYBPC; MYBPC3; Myosin binding protein C cardiac; Myosin binding protein C cardiac-type; Myosin-binding protein C; MYPC3_HUMAN.
Organism Species:	Rabbit
Clonality:	Polyclonal
React Species:	Human,Mouse,Rat,Cow,Horse,Rabbit,
Applications:	WB=1:500-2000ELISA=1:500-1000IHC-P=1:400-800IHC-F=1:400-800IF=1:50-200 (Paraffin sections need antigen repair) not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight:	141kDa
Cellular localization:	cytoplasmic
Form:	Lyophilized or Liquid
Concentration:	1mg/ml
immunogen:	KLH conjugated synthetic peptide derived from human MYBPC3:161-260/1274
Lsotype:	IgG
Purification:	affinity purified by Protein A
Storage Buffer:	0.01M TBS(pH7.4) with 1% BSA, 0.03% Proclin300 and 50% Glycerol.
Storage:	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.
PubMed:	PubMed
Product Detail:	MYBPC3 encodes the cardiac isoform of the thick-filament myosin-binding protein C. It is found in the crossbridge-bearing zone (C region) of A bands in vertebrate striated muscle. Regulatory phosphorylation of MYBPC3 by cAMP-dependent protein kinase (PKA) upon adrenergic stimulation may be linked to modulation of cardiac contraction.

MYBPC3 binds F-Actin, MHC and native thin filaments, and modifies the activity of Actin-activated myosin ATPase. Mutations in the MYBPC3 gene lead mainly to truncation of the protein, which results in one cause of familial hypertrophic cardiomyopathy type 4 (CMH4), a heart disorder characterized by ventricular hypertrophy, which often involves the interventricular septum and is usually asymmetric. The MYBPC3 gene maps to chromosome 11p11.2.

Function:

Thick filament-associated protein located in the crossbridge region of vertebrate striated muscle a bands. In vitro it binds MHC, F-actin and native thin filaments, and modifies the activity of actin-activated myosin ATPase. It may modulate muscle contraction or may play a more structural role.

Post-translational modifications:

Substrate for phosphorylation by PKA and PKC. Reversible phosphorylation appears to modulate contraction (By similarity).

DISEASE:

Defects in MYBPC3 are the cause of familial hypertrophic cardiomyopathy type 4 (CMH4) [MIM:115197]. 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.

Similarity:

Belongs to the immunoglobulin superfamily. MyBP family.
Contains 3 fibronectin type-III domains.
Contains 7 Ig-like C2-type (immunoglobulin-like) domains.

SWISS:

Q14896

Gene ID:

4607

Database links:

[Entrez Gene: 4607](#)Human

[Entrez Gene: 17868](#)Mouse

[Entrez Gene: 295929](#)Rat

[Omim: 600958](#)Human

[SwissProt: Q14896](#)Human

[SwissProt: O70468](#)Mouse

[SwissProt: P56741](#)Rat

[Unigene: 524906](#)Human

[Unigene: 10728](#)Mouse

[Unigene: 162668](#)Rat

Important Note:

This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.

Involvement in disease: Defects in MYBPC3 are the cause of cardiomyopathy familial hypertrophic type 4 (CMH4). 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.

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