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Rabbit Anti-MYBPC3/Cy5 Conjugated antibody

SL9868R-Cy5

Product Name	Anti-MYBPC3/Cy5
Chinese Name	Cy5 标记的心脏肌球蛋白 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.
Research Area	Cardiovascular immunology
Immunogen Species	Rabbit
Clonality	Polyclonal
React Species	(predicted:Human,Mouse,Rat,Cow,Horse,Rabbit)
Applications	IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Molecular weight	141kDa
Form	Lyophilized or Liquid
Concentration	1mg/ml
immunogen	KLH conjugated synthetic peptide derived from human MYBPC3
Lsotype	IgG
Purification	affinity purified by Protein A
Storage Buffer	1M TBS(pH7.4) with 1% BSA, 3% 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 1M PBS or diluent of antibody the antibody is stable for at least two weeks at 2-4 °C.
Product Detail	**background:** 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.

Database links:

Entrez Gene: 4607 Human

Entrez Gene: 17868 Mouse

Entrez Gene: 295929 Rat

Omim: 600958 Human

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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.