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[Marker Primary Antibody]

Rabbit Anti-MYBPC3/PE-Cy7 Conjugated antibody

Catalog Number: SL9868R-PE-Cy7

FOR RESEARCH USE ONLY

[DATASHEET]

Host:	Rabbit
Isotype:	IgG
Clonality:	Polyclonal
Target:	Anti-MYBPC3/PE-Cy7
Immunogen:	KLH conjugated synthetic peptide derived from human MYBPC3
Purification:	affinity purified by Protein A
Concentration:	1mg/ml
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.
Applications:	IF=1:100-500 not yet tested in other applications. optimal dilutions/concentrations should be determined by the end user.
Reactivity:	(predicted:Human,Mouse,Rat,Cow,Horse,Rabbit)
Background:	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.

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

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	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 **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.