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Ryanodine Receptor Polyclonal Antibody
Purified Rabbit Polyclonal Antibody (Pab)
Catalog # AP58415

Specification

Ryanodine Receptor Polyclonal Antibody - Product Information

Application	**IHC-P, IHC-F, IF, E**
Primary Accession	P21817, Q92736, Q15413
Reactivity	**Rat, Pig, Dog, Bovine**
Host	**Rabbit**
Clonality	**Polyclonal**
Calculated MW	**566 KDa**
Physical State	**Liquid**
Immunogen	**KLH conjugated synthetic peptide derived from human Ryanodine Receptor**
Epitope Specificity	**4701-4800/5038**
Isotype	**IgG**
Purity affinity purified by Protein A	
Buffer	**0.01M TBS (pH7.4) with 1% BSA, 0.02% Proclin300 and 50% Glycerol.**
SUBCELLULAR LOCATION	**Sarcoplasmic reticulum membrane; Multi-pass membrane protein (Probable). Membrane; Multi-pass membrane protein. Microsome membrane; Multi-pass membrane protein.**
SIMILARITY	**Belongs to the ryanodine receptor (TC 1.A.3.1) family. RYR3 subfamily.Contains 3 B30.2/SPRY domains.Contains 5 MIR domains.**
SUBUNIT	**Homotetramer. Can also form heterotetramers with RYR2.Interacts with CALM; CALM with bound calcium inhibits the RYR1channel activity. Interacts with S100A1. Interacts with FKBP1A;this stabilizes the closed conformation of the channel. Interactswith CACNA1S; interaction with CACNA1S is important for activationof the RYR1 channel. Interacts with CACNB1. Interacts with TRDN andASPH; these interactions stimulate RYR1 channel activity (Bysimilarity). Identified in a complex composed of RYR1, PDE4D, PKA,FKBP1A and protein phosphatase 1 (PP1). Repeated very high-levelexercise decreases interaction with PDE4D and protein phosphatase 1(PP1).**
Post-translational modifications	**Channel activity is modulated by phosphorylation.Phosphorylation at**

	Ser-2843 may increase channel activity. Repeatedvery high-level exercise increases phosphorylation at Ser-2843.[PTM] Activated by reversible S-nitrosylation. Repeated veryhigh-level exercise increases S-nitrosylation.
DISEASE	**Malignant hyperthermia 1 (MHS1) [MIM:145600]: Autosomaldominant pharmacogenetic disorder of skeletal muscle and is one ofthe main causes of death due to anesthesia. In susceptible people,an MH episode can be triggered by all commonly used inhalationalanesthetics such as halothane and by depolarizing muscle relaxantssuch as succinylcholine. The clinical features of the myopathy arehyperthermia, accelerated muscle metabolism, contractures,metabolic acidosis, tachycardia and death, if not treated with thepostsynaptic muscle relaxant, dantrolene. Susceptibility to MH canbe determined with the 'in vitro' contracture test (IVCT):observing the magnitude of contractures induced in strips of muscletissue by caffeine alone and halothane alone. Patients with normalresponse are MH normal (MHN), those with abnormal response tocaffeine alone or halothane alone are MH equivocal (MHE(C) andMHE(H) respectively). Note=The disease is caused by mutationsaffecting the gene represented in this entry.Central core disease of muscle (CCD) [MIM:117000]:Autosomal dominant congenital myopathy, but a severe autosomalrecessive form also exists. Both clinical and histologicalvariability is observed. Affected individuals typically displayhypotonia and proximal muscle weakness in infancy, leading to thedelay of motor milestones. The clinical course of the disorder isusually slow or nonprogressive in adulthood, and the severity ofthe symptoms may vary from normal to significant muscle weakness.Microscopic examination of CCD-affected skeletal muscle reveals apredominance of type I fibers containing amorphous-looking areas(cores) that do not stain with oxidative and phosphorylasehistochemical techniques. Note=The disease is caused by mutationsaffecting the gene represented in this entry. Multiminicore disease with external ophthalmoplegia(MMDO) [MIM:255320]: Clinically heterogeneous neuromusculardisorder. General features**

	include neonatal hypotonia, delayedmotor development, and generalized muscle weakness and amyotrophy,which may progress slowly or remain stable. Muscle biopsy showsmultiple, poorly circumscribed, short areas of sarcomeredisorganization and mitochondria depletion (areas termed minicores)in most muscle fibers. Typically, no dystrophic signs, such asmuscle fiber necrosis or regeneration or significant endomysialfibrosis, are present in multiminicore disease. Note=The disease iscaused by mutations affecting the gene represented in this entry. Congenital myopathy with fiber-type disproportion (CFTD)[MIM:255310]: Genetically heterogeneous disorder in which there isrelative hypotrophy of type 1 muscle fibers compared to type 2fibers on skeletal muscle biopsy. However, these findings are notspecific and can be found in many different myopathic andneuropathic conditions. Note=The disease is caused by mutationsaffecting the gene represented in this entry. Note=Defects in RYR1 may be a cause of Samaritanmyopathy, a congenital myopathy with benign course. Patientsdisplay severe hypotonia and respiratory distress at birth. Unlikeother congenital myopathies, the health status constantly improvesand patients are minimally affected at adulthood.
Important Note	**This product as supplied is intended for research use only, not for use in human, therapeutic or diagnostic applications.**

Background Descriptions

The Ryanodine Receptor (RyR) is the channel responsible for calcium release from muscle cell Sarcoplasmic Reticulum (SR) and also plays a role in calcium regulation in non-muscle cells. The RyR exists as a homotetramer and is predicted to have a short cytoplasmic C-terminus and 4-10 transmembrane domains. The remainder of the protein, termed the "foot" region, is located in the cytoplasm between the transverse tubule and the SR. Mammalian RyR isoforms are the product of three different genes: RyR-1 is expressed predominantly in skeletal muscle and areas of the brain; RyR-2 is expressed predominantly in heart muscle but also found in the stomach, endothelial cells and diffuse areas of the brain; and RyR-3 is found in smooth muscle and the brain (striatum, thalamus and hippocampus). In non-mammalian vertebrates, the RyR isoforms are termed alpha, beta and cardiac which correlate loosely to the mammalian RyR-1, RyR-3 and RyR-2 isoforms respectively.

Ryanodine Receptor Polyclonal Antibody - Additional Information

Target/Specificity

Brain, skeletal muscle, placenta and possibly liver and kidney. In brain, highest levels are found in the cerebellum, hippocampus, caudate nucleus and amygdala, with lower levels in the corpus callosum, substantia nigra and thalamus.

Dilution
IHC-P~~N/A<br \>IHC-F~~N/A<br \>IF~~1:50~200<br \>E~~N/A

Format
0.01M TBS(pH7.4), 0.09% (W/V) sodium azide and 50% Glyce

Storage
Store at -20 ℃ for one year. Avoid repeated freeze/thaw cycles. 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 ℃.

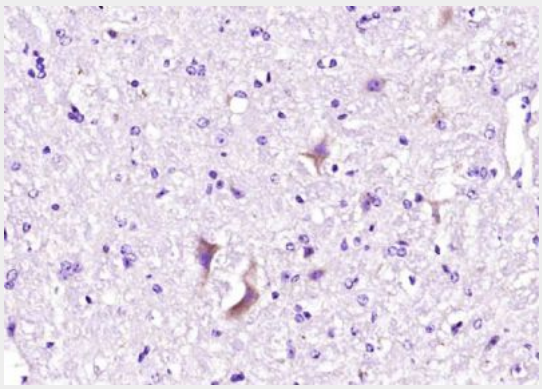
Ryanodine Receptor Polyclonal Antibody - Protein Information

Ryanodine Receptor Polyclonal Antibody - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Western Blot
- Blocking Peptides
- Dot Blot
- Immunohistochemistry
- Immunofluorescence
- Immunoprecipitation
- Flow Cytomety
- Cell Culture

Ryanodine Receptor Polyclonal Antibody - Images



Paraformaldehyde-fixed, paraffin embedded (mouse cerebellum tissue); Antigen retrieval by boiling in sodium citrate buffer (pH6.0) for 15min; Block endogenous peroxidase by 3% hydrogen peroxide for 20 minutes; Blocking buffer (normal goat serum) at 37°C for 30min; Antibody incubation with (Ryanodine Receptor) Polyclonal Antibody, Unconjugated (bs-6305R) at 1:400 overnight at 4°C, followed by operating according to SP Kit(Rabbit) (sp-0023) instructionsand DAB staining.