SANTA CRUZ BIOTECHNOLOGY, INC.

RyR (C-18): sc-8169

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

Dihydropyridine receptor (DHPR) is a surface membrane protein critical for the excitation-contraction coupling of striated muscle. DHPR and the sarcoplasmic reticulum ryanodine receptor (RyR) are two key components of the intracellular junctions, where depolarization of the surface membrane is converted into the release of Ca^{2+} from internal stores. The α1-subunit of the DHPR contains a cytoplasmic loop which is thought to be involved in the interactions with RyR. Phosphorylation of the DHPR α1-subunit is also thought to play a role in the functional interaction of DHPR and RyR. Mutation in DHPR α1 results in excitation-contraction uncoupling, leading to muscular dysgenesis, a complete inactivity in developing skeletal muscles. Cells that do not express RyR also lack excitation-contraction coupling and exhibit a several-fold reduction in Ca^{2+} current density.

REFERENCES

1. Pincon-Raymond, M., et al. 1990. A genetic model for the study of abnormal nerve-muscle interactions at the level of excitation-contraction coupling: the mutation muscular dysgenesis. J. Physiol. 84: 82-87.
2. Fan, H., et al. 1995. Binding sites of monoclonal antibodies and dihydropyridine receptor α1 subunit cytoplasmic II-III loop on skeletal muscle triadin fusion peptides. Biochemistry 34: 14893-14901.
3. Lu, X., et al. 1995. Phosphorylation of dihydropyridine receptor II-III loop peptide regulates skeletal muscle calcium release channel function. Evidence for an essential role of the β-OH group of Ser687. J. Biol. Chem. 270: 18459-18464.
4. Powell, J.A., et al. 1996. Formation of triads without the dihydropyridine receptor α subunits in cell lines from dysgenic skeletal muscle. J. Cell Biol. 134: 375-387.
5. Flucher, B.E., et al. 1996. Formation of junctions involved in excitation-contraction coupling in skeletal and cardiac muscle. Proc. Natl. Acad. Sci. USA 93: 8101-8106.
6. Franzini-Armstrong, C., et al. 1997. Ryanodine receptors of striated muscles: a complex channel capable of multiple interactions. Physiol. Rev. 77: 699-729.

SOURCE

RyR (C-18) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of RyR of human origin.

PRODUCT

Each vial contains 200 µg IgG in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Blocking peptide available for competition studies, sc-8169 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

STORAGE

Store at 4° C, **DO NOT FREEZE**. Stable for one year from the date of shipment. Non-hazardous. No MSDS required.

APPLICATIONS

RyR (C-18) is recommended for detection of skeletal muscle, cardiac muscle and brain ryanodine receptors of mouse, rat and human origin by immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

RyR (C-18) is also recommended for detection of skeletal muscle, cardiac muscle and brain ryanodine receptors in additional species, including equine, canine, bovine, porcine and avian.

Molecular Weight of RyR-1: 550 kDa.

Molecular Weight of RyR-2: 565 kDa.

Molecular Weight of RyR-3: 552 kDa.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Immunofluorescence: use donkey anti-goat IgG-FITC: sc-2024 (dilution range: 1:100-1:400) or donkey anti-goat IgG-TR: sc-2783 (dilution range: 1:100-1:400) with UltraCruz™ Mounting Medium: sc-24941.

SELECT PRODUCT CITATIONS

1. Guse, A.H., et al. 2001. Transient tyrosine phosphorylation of human ryanodine receptor upon T cell stimulation. J. Biol. Chem. 276: 34722-34727.
2. Balakier, H., et al. 2002. Calcium-binding proteins and calcium-release channels in human maturing oocytes, pronuclear zygotes and early preimplantation embryos. Hum. Reprod. 17: 2938-2947.
3. Panfoli, I., et al. 2007. Localization of the cyclic ADP-ribose-dependent calcium signaling pathway in bovine rod outer segments. Invest. Ophthalmol. Vis. Sci. 48: 978-984.
4. Costa, R.R., et al. 2010. A calcium-induced calcium release mechanism supports luteinizing hormone-induced testosterone secretion in mouse Leydig cells. Am. J. Physiol., Cell Physiol. 299: C316-C323.

RESEARCH USE

For research use only, not for use in diagnostic procedures.

PROTOCOLS

See our web site at www.scbt.com or our catalog for detailed protocols and support products.