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Product Information

Anti-Calsequestrin-1 (N-terminal)
produced in rabbit, affinity isolated antibody

Catalog Number **C0743**

Product Description

Anti-Calsequestrin-1 (N-terminal) is produced in rabbit using as immunogen a synthetic peptide corresponding to amino acids 28-45 located near the N-terminus of mouse calsequestrin-1 (GeneID: 12372), conjugated to KLH. This sequence is identical in rat calsequestrin-1 and highly conserved in human and bovine calsequestrin-1 (two amino acid substitution). This sequence is not found in calsequestrin-2. The antibody is affinity purified using the immunizing peptide immobilized on agarose.

Anti-Calsequestrin-1 (N-terminal) specifically recognizes calsequestrin-1 by immunoblotting (~60 kDa). Staining of the calsequestrin-1 band in immunoblotting is specifically inhibited by the immunizing peptide (mouse, amino acids 28-45).

Calcium (Ca^{2+}) plays an important role as a messenger in the excitation-contraction coupling process of muscle cells. It is stored in the sarcoplasmic reticulum (SR) and released via the Ca^{2+} release channel/ryanodine receptor (RyR) complex. The Ca^{2+} concentration in muscle undergoes rhythmic changes due in large part to the Ca^{2+} transport and storage properties of the SR, which controls the state of contraction of the myofiber. Ca^{2+} is actively transported into the SR by the Ca^{2+}-dependent ATPases (SERCAs), transiently stored by the Ca^{2+}-binding protein calsequestrin and subsequently released by inositol 1,4,5-trisphosphate via the Ca^{2+} release channel/ryanodine receptor (RyR) resulting in muscle contraction.

Calsequestrin (CS also known as CSQ), the major Ca^{2+} binding protein in cardiac and skeletal muscle, is a high-capacity, low-affinity Ca^{2+} binding glycoprotein, which functions as an internal Ca^{2+} store in the lumen of the SR.[1-3] In mammals, two forms of the protein exist, calsequestrin-1 (CASQ-1, calmitin, aspartactin, laminin-binding protein) and calsequestrin-2 (CASQ-2), which encode the fast-twitch skeletal muscle and cardiac calsequestrin, respectively. Calsequestrin-1 (60 kDa) is found in the SR's terminal cisternae luminal space of fast skeletal muscle cells.[1,2,4,5]

Calsequestrin-1 has been identified as a putative autoantigen associated with eye muscle inflammation in Graves' disease.[6,7] Calsequestrin-2 (55 kDa) is found in the SR's terminal cisternae luminal space of both cardiac and slow skeletal muscle cells.[3,8] Mutations in CASQ2 are thought to be associated with catecholaminergic polymorphic ventricular tachycardia (CPVT). Calsequestrin is thought to regulate RyR signaling and Ca^{2+} release through triadin and junctin proteins.[11,12]

Reagent

Supplied as a solution in 0.01 M phosphate buffered saline, pH 7.4, containing 15 mM sodium azide as a preservative.

Antibody concentration: ~1.0 mg/mL

Precautions and Disclaimer

This product is for R&D use only, not for drug, household, or other uses. Please consult the Material Safety Data Sheet for information regarding hazards and safe handling practices.

Storage/Stability

For extended storage, freeze in working aliquots at –20 °C. Repeated freezing and thawing, or storage in "frost-free" freezers, is not recommended. If slight turbidity occurs upon prolonged storage, clarify the solution by centrifugation before use. Working dilution samples should be discarded if not used within 12 hours. For continuous use, store at 2–8 °C for up to one month.

Product Profile

Immunoblotting: a working antibody concentration of 0.5-1.0 μg/mL is recommended using an extract of rat skeletal muscle S1 fraction.

Note: In order to obtain the best results and assay sensitivity in various techniques and preparations, we recommend determining optimal working concentrations by titration.

References

1. McLennan, D.H., and Wong, P.T.S., *Proc. Natl. Acad. Sci. USA*, **68**, 1231-1235 (1971).
2. Jorgensen, A.O., et al., *J. Cell Biol.*, **80**, 372-384 (1979).
3. Jorgensen, A.O., et al., *J. Cell Biol.*, **98**, 1597-1602 (1984).
4. Nori, A., et al., *Exp. Cell Res.*, **260**, 40-49 (2000).
5. Nori, A., et al., *Am. J. Physiol. Cell Physiol.*, **291**, C245-C253 (2006).
6. Gunji, K., et al., *Autoimmunity*, **29**, 1-9 (1999).
7. Gopinath, B., et al., *Clin. Exp. Immunol.*, **145**, 56-62 (2006).
8. Volpe, P., et al., *Biochem. J.*, **301**, 465-469 (1994).
9. di Barletta, M.R., et al., *Circulation*, **114**, 1012-1019 (2006).
10. Viatchenko-Karpinski, S., et al., *Circ. Res.*, **94**, 471-477 (2004).
11. Lee, J.M., et al., *J. Biol. Chem.*, **279**, 6994-7000 (2004).
12. Györke, I., et al., *Biophys. J.*, **86**, 2121-2128 (2004).

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