SANTA CRUZ BIOTECHNOLOGY, INC.

γ-sarcoglycan (K-17): sc-14185

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The sarcoglycan transmembrane proteins are members of the dystrophin complex. Sarcoglycans cluster together to form a complex, which is localized in the cell membrane of skeletal, cardiac, and smooth muscle fibers. Four sarcoglycan subunit proteins, designated α-, β-, γ- and δ-sarcoglycan, form a complex on the skeletal muscle cell surface membrane. A genetic defect in any one of these proteins causes the loss or marked decrease of the whole sarcoglycan complex, which is observed in the autosomal recessive muscular dystrophy, sarcoglycanopathy. In smooth muscle, β- and δ-sarcoglycans are associated with ε-sarcoglycan, a glycoprotein homologous to α-sarcoglycan. Additionally, a complete deficiency in δ-sarcoglycan is the cause of the Syrian hamster BIO.14 cardiomyopathy.

REFERENCES

1. Barresi, R., et al. 2000. Expression of γ-sarcoglycan in smooth muscle and its interaction with the smooth muscle sarcoglycan-sarcospan complex. J. Biol. Chem. 275: 38554-38560.
2. Hack, A.A., et al. 2000. Differential requirement for individual sarcoglycans and dystrophin in the assembly and function of the dystrophin-glycoprotein complex. J. Cell Sci. 113: 2535-2544.
3. Enigk, R.E., et al. 2001. Cellular and molecular properties of α-dystrobrevin in skeletal muscle. Front. Biosci. 6: D 53-64.
4. Politano, L., et al. 2001. Evaluation of cardiac and respiratory involvement in sarcoglycanopathies. Neuromuscul. Disord. 11: 178-185.
5. Ueda, H., et al. 2001. δ- and γ-sarcoglycan localization in the sarcoplasmic reticulum of skeletal muscle. J. Histochem. Cytochem. 49: 529-538.
6. Wakabayashi-Takai, E., et al. 2001. Identification of myogenesis-dependent transcriptional enhancers in promoter region of mouse γ-sarcoglycan gene. Eur. J. Biochem. 268: 948-957.
7. Anastasi, G., et al. 2004. Sarcoglycan and integrin localization in normal human skeletal muscle: a confocal laser scanning microscope study. Eur. J. Histochem. 48: 245-252.
8. Lapidos, K.A., et al. 2004. Transplanted hematopoietic stem cells demonstrate impaired sarcoglycan expression after engraftment into cardiac and skeletal muscle. J. Clin. Invest. 114: 1577-1585.

CHROMOSOMAL LOCATION

Genetic locus: Sgcg (mouse) mapping to 14 D1.

SOURCE

γ-sarcoglycan (K-17) is an affinity purified goat polyclonal antibody raised against a peptide mapping near the C-terminus of γ-sarcoglycan of mouse origin.

STORAGE

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

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-14185 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

γ-sarcoglycan (K-17) is recommended for detection of γ-sarcoglycan of mouse and rat origin by Western Blotting (starting dilution 1:200, dilution range 1:100-1:1000), 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).

Suitable for use as control antibody for γ-sarcoglycan siRNA (m): sc-43425, γ-sarcoglycan shRNA Plasmid (m): sc-43425-SH and γ-sarcoglycan shRNA (m) Lentiviral Particles: sc-43425-V.

Molecular Weight of γ-sarcoglycan: 35 kDa.

RECOMMENDED SECONDARY REAGENTS

To ensure optimal results, the following support (secondary) reagents are recommended: 1) Western Blotting: use donkey anti-goat IgG-HRP: sc-2020 (dilution range: 1:2000-1:100,000) or Cruz Marker™ compatible donkey anti-goat IgG-HRP: sc-2033 (dilution range: 1:2000-1:5000), Cruz Marker™ Molecular Weight Standards: sc-2035, TBS Blotto A Blocking Reagent: sc-2333 and Western Blotting Luminol Reagent: sc-2048. 2) 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. Blanco, G., et al. 2004. Molecular phenotyping of the mouse ky mutant reveals UCP1 upregulation at the neuromuscular junctions of dystrophic soleus muscle. Neuromuscul. Disord. 14: 217-228.

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.