SANTA CRUZ BIOTECHNOLOGY, INC.

Tafazzin (F-7): sc-365810

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The Power to Question

BACKGROUND

Tafazzin protein is a single-pass membrane protein that is abundant in cardiac and skeletal muscle, where it influences mitochondrial structure. There are various isoforms associated with Tafazzin, most of which are ubiquitous. Isoforms with hydrophobic N-terminal domains are membrane anchored, whereas the short isoforms that lack a hydrophobic leader sequence may exist as cytoplasmic proteins. The isoforms that lack the N-terminal domain are not found in cardiac or skeletal muscle, rather they are located in fibroblasts and leukocytes. Mutations in the Tafazzin gene are associated with various diseases, including dilated cardiomyopathy (DCM), hypertrophic DCM, endocardial fibroelastosis, left ventricular noncompaction (LVNC) and Barth syndrome (BTHS), a severe inherited disorder marked by neutropenia, cardiac and skeletal myopathy and short stature.

REFERENCES

1. Schlame, M., et al. 2003. Phospholipid abnormalities in children with Barth syndrome. J. Am. Coll. Cardiol. 42: 1994-1999.
2. Gu, Z., et al. 2004. Aberrant cardiolipin metabolism in the yeast taz1 mutant: a model for Barth syndrome. Mol. Microbiol. 51: 149-158.
3. Lu, B., et al. 2004. Complex expression pattern of the Barth syndrome gene product Tafazzin in human cell lines and murine tissues. Biochem. Cell Biol. 82: 569-576.

CHROMOSOMAL LOCATION

Genetic locus: TAZ (human) mapping to Xq28; Taz (mouse) mapping to X A7.3.

SOURCE

Tafazzin (F-7) is a mouse monoclonal antibody specific for an epitope mapping between amino 209-241 near the C-terminus of Tafazzin of human origin.

PRODUCT

Each vial contains 200 µg IgG_{2b} kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

Tafazzin (F-7) is available conjugated to agarose (sc-365810 AC), 500 µg/ 0.25 ml agarose in 1 ml, for IP; to HRP (sc-365810 HRP), 200 µg/ml, for WB, IHC(P) and ELISA; to either phycoerythrin (sc-365810 PE), fluorescein (sc-365810 FITC), Alexa Fluor® 488 (sc-365810 AF488), Alexa Fluor® 546 (sc-365810 AF546), Alexa Fluor® 594 (sc-365810 AF594) or Alexa Fluor® 647 (sc-365810 AF647), 200 µg/ml, for WB (RGB), IF, IHC(P) and FCM; and to either Alexa Fluor® 680 (sc-365810 AF680) or Alexa Fluor® 790 (sc-365810 AF790), 200 µg/ml, for Near-Infrared (NIR) WB, IF and FCM.

Blocking peptide available for competition studies, sc-365810 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% stabilizer protein).

STORAGE

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

APPLICATIONS

Tafazzin (F-7) is recommended for detection of all isoforms of Tafazzin of mouse, rat and human origin by Western Blotting (starting dilution 1:100, dilution range 1:100-1:1000), immunoprecipitation [1-2 µg per 100-500 µg of total protein (1 ml of cell lysate)], immunofluorescence (starting dilution 1:50, dilution range 1:50-1:500), immunohistochemistry (including paraffin-embedded sections) (starting dilution 1:50, dilution range 1:50-1:500) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

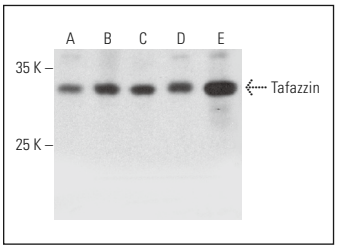
Tafazzin (F-7) is also recommended for detection of all isoforms of Tafazzin in additional species, including equine, canine and bovine.

Suitable for use as control antibody for Tafazzin siRNA (h): sc-61637, Tafazzin siRNA (m): sc-61638, Tafazzin shRNA Plasmid (h): sc-61637-SH, Tafazzin shRNA Plasmid (m): sc-61638-SH, Tafazzin shRNA (h) Lentiviral Particles: sc-61637-V and Tafazzin shRNA (m) Lentiviral Particles: sc-61638-V.

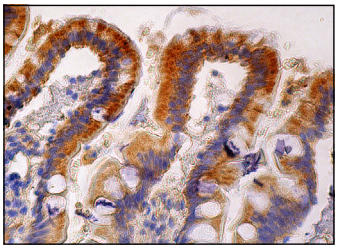
Molecular Weight of Tafazzin: 34 kDa.

Positive Controls: NIH/3T3 whole cell lysate: sc-2210, RAW 264.7 whole cell lysate: sc-2211 or SW-13 cell lysate: sc-24778.

DATA



Tafazzin (F-7): sc-365810. Western blot analysis of Tafazzin expression in SW-13 (**A**), NIH/3T3 (**B**), RAW 264.7 (**C**), NRK (**D**) and RIN-m5F (**E**) whole cell lysates.



Tafazzin (F-7): sc-365810. Immunoperoxidase staining of formalin fixed, paraffin-embedded human small intestine tissue showing cytoplasmic staining of glandular cells.

SELECT PRODUCT CITATIONS

1. Chao, H., et al. 2018. Disentangling oxidation/hydrolysis reactions of brain mitochondrial cardiolipins in pathogenesis of traumatic injury. JCI Insight 3: e97677.
2. Le, C.H., et al. 2020. Tafazzin deficiency impairs CoA-dependent oxidative metabolism in cardiac mitochondria. J. Biol. Chem. 295: 12485-12497.
3. Zegallai, H.M., et al. 2021. Tafazzin deficiency impairs mitochondrial metabolism and function of lipopolysaccharide activated B lymphocytes in mice. FASEB J. 35: e22023.
4. Zhou, M., et al. 2022. Generation of a homozygous TAZ knockout hESCs line by CRISPR/Cas9 system. Stem Cell Res. 64: 102923.
5. Liu, L.X., et al. 2024. SIRT3 regulates cardiolipin biosynthesis in pressure overload-induced cardiac remodeling by PPARγ-mediated mechanism. PLoS ONE 19: e0301990.

RESEARCH USE

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