SANTA CRUZ BIOTECHNOLOGY, INC.

SQSTM1 (H-290): sc-25575

SANTA CRUZ BIOTECHNOLOGY

The Power to Question

BACKGROUND

The chronic focal skeletal disorder, Paget's disease of bone, affects 2-3% of the population over the age of 60 years. Paget's disease is characterized by increased bone resorption by osteoclasts, followed by abundant new bone formation that is of poor quality. The disease leads to several complications including bone pain and deformities, as well as fissures and fractures. Mutations in the ubiquitin-associated (UBA) domain of the sequestosome 1 protein (SQSTM1, also designated p62 or ZIP) commonly cause Paget's disease since the UBA is necessary for aggregate sequestration and cell survival.

REFERENCES

1. Hocking, L.J., et al. 2002. Domain-specific mutations in sequestosome 1 (SQSTM1) cause familial and sporadic Paget's disease. Hum. Mol. Genet. 11: 2735-2739.
2. Rousiere, M., et al. 2003. Paget's disease of bone. Best Pract. Res. Clin. Rheumatol. 17: 1019-1041.

CHROMOSOMAL LOCATION

Genetic locus: SQSTM1 (human) mapping to 5q35.3; Sqstm1 (mouse) mapping to 11 B1.3.

SOURCE

SQSTM1 (H-290) is a rabbit polyclonal antibody raised against amino acids 151-440 of SQSTM1 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.

APPLICATIONS

SQSTM1 (H-290) is recommended for detection of sequestosome 1 of mouse, rat and human origin by Western Blotting (starting dilution 1:200, 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) and solid phase ELISA (starting dilution 1:30, dilution range 1:30-1:3000).

SQSTM1 (H-290) is also recommended for detection of sequestosome 1 in additional species, including equine, bovine and porcine.

Suitable for use as control antibody for SQSTM1 siRNA (h): sc-29679, SQSTM1 siRNA (m): sc-29828, SQSTM1 shRNA Plasmid (h): sc-29679-SH, SQSTM1 shRNA Plasmid (m): sc-29828-SH, SQSTM1 shRNA (h) Lentiviral Particles: sc-29679-V and SQSTM1 shRNA (m) Lentiviral Particles: sc-29828-V.

Molecular Weight of SQSTM1: 65 kDa.

Positive Controls: SK-BR-3 cell lysate: sc-2218, HeLa whole cell lysate: sc-2200 or SK-LMS-1 cell lysate: sc-3813.

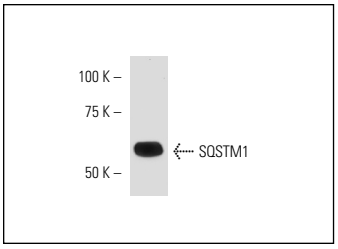
STORAGE

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

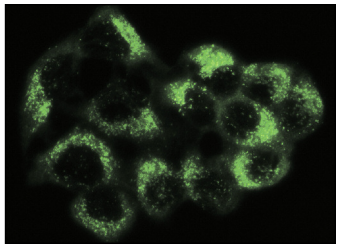
RESEARCH USE

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

DATA



SQSTM1 (H-290): sc-25575. Western blot analysis of SQSTM1 expression in SK-LMS-1 whole cell lysate.



SQSTM1 (H-290): sc-25575. Immunofluorescence staining of methanol-fixed HeLa cells showing cytoplasmic localization.

SELECT PRODUCT CITATIONS

1. Nogalska, A., et al. 2009. p62/SQSTM1 is overexpressed and prominently accumulated in inclusions of sporadic inclusion-body myositis muscle fibers, and can help differentiating it from polymyositis and dermatomyositis. Acta Neuropathol. 118: 407-413.
2. Colosetti, P., et al. 2009. Autophagy is an important event for megakaryocytic differentiation of the chronic myelogenous leukemia K562 cell line. Autophagy 5: 1092-1098.
3. Gal, J., et al. 2009. Sequestosome 1/p62 links familial ALS mutant SOD1 to LC3 via an ubiquitin-independent mechanism. J. Neurochem. 111: 1062-1073.
4. Jiang, H., et al. 2009. Autophagy pathways in glioblastoma. Methods Enzymol. 453: 273-286.
5. Inomata, M., et al. 2012. Regulation of Toll-like receptor signaling by NDP52-mediated selective autophagy is normally inactivated by A20. Cell. Mol. Life Sci. 69: 963-979.
6. Lee, H.M., et al. 2012. *Mycobacterium abscessus* activates the NLRP3 inflammasome via Dectin-1-Syk and p62/SQSTM1. Immunol. Cell Biol. 90: 601-610.
7. Zheng, X., et al. 2012. Effect of p62 on Tau hyperphosphorylation in a rat model of Alzheimer's disease. Neural Regen. Res. 7: 1304-1311.
8. Belaid, A., et al. 2013. Autophagy plays a critical role in the degradation of active RHOA, the control of cell cytokinesis and genomic stability. Cancer Res. 73: 4311-4322.
9. Baldelli, S., et al. 2014. PGC-1α buffers ROS-mediated removal of mitochondria during myogenesis. Cell Death Dis. 5: e1515.