SANTA CRUZ BIOTECHNOLOGY, INC.

Menin (C-19): sc-8200

BACKGROUND

Menin (multiple endocrine neoplasia I, MEN1, MEAI, SCG2) is a nuclear tumor suppressor that is mutated in patients with multiple endocrine neoplasia type I (MEN1). Menin can activate the transcription of differentiation-regulating genes by covalent histone modification. In osteoblasts, the interaction of menin and the TGFβ/Smad3 pathway negatively regulates BMP2/Smad1/5- and Runx2-dependent transcription activities leading to inhibition of late-stage differentiation. Menin regulates the expression of IGFBP-2 by influencing the IGFBP-2 promoter. Ectopic overexpression of menin via adenoviruses induces apoptosis in murine embryonic fibroblasts in a Bax/Bak-dependent manner. Two mRNA exist and two variants of the shorter mRNA have alternative splicing that changes the CDS. Five variants where alternative splicing takes place in the 5' UTR have been identified.

CHROMOSOMAL LOCATION

Genetic locus: MEN1 (human) mapping to 11q13.1; Men1 (mouse) mapping to 19 A.

SOURCE

Menin (C-19) is an affinity purified goat polyclonal antibody raised against a peptide mapping at the C-terminus of Menin 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.

Menin (C-19) is available conjugated phycoerythrin (sc-8200 PE, 200 µg/ml), for IF, IHC(P) and FCM.

Blocking peptide available for competition studies, sc-8200 P, (100 µg peptide in 0.5 ml PBS containing < 0.1% sodium azide and 0.2% BSA).

APPLICATIONS

Menin (C-19) is recommended for detection of Menin 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)].

Menin (C-19) is also recommended for detection of Menin in additional species, including equine, canine, bovine and porcine.

Suitable for use as control antibody for Menin siRNA (h): sc-35922, Menin siRNA (m): sc-35923, Menin shRNA Plasmid (h): sc-35922-SH, Menin shRNA Plasmid (m): sc-35923-SH, Menin shRNA (h) Lentiviral Particles: sc-35922-V and Menin shRNA (m) Lentiviral Particles: sc-35923-V.

Molecular Weight of Menin: 67 kDa.

Positive Controls: K-562 nuclear extract: sc-2130, Jurkat + PMA nuclear extract: sc-2133 or A-431 nuclear extract: sc-2122.

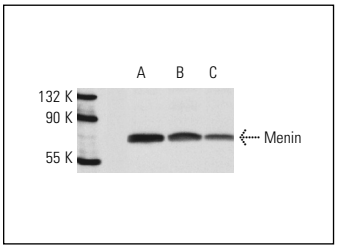
STORAGE

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

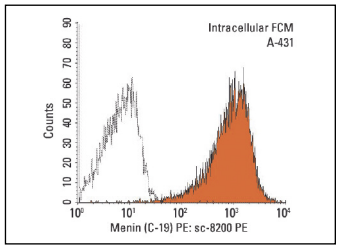
RESEARCH USE

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

DATA



Menin (C-19): sc-8200. Western blot analysis of Menin expression in phorbol-induced Jurkat (**A**), K-562 (**B**) and A-431 (**C**) nuclear extracts.



Menin (C-19) PE: sc-8200 PE. Intracellular FCM analysis of fixed and permeabilized A431 cells. Black line histogram represents the isotype control, normal goat IgG: sc-3992.

SELECT PRODUCT CITATIONS

1. Scacheri, P.C., et al. 2001. Bidirectional transcriptional activity of PGK-neomycin and unexpected embryonic lethality in heterozygote chimeric knockout mice. Genesis 30: 259-263.
2. Sowa, H., et al. 2004. Menin inactivation leads to loss of transforming growth factor β inhibition of parathyroid cell proliferation and parathyroid hormone secretion. Cancer Res. 64: 2222-2228.
3. Kidd, M., et al. 2004. Global expression analysis of ECL cells in *Mastomys natalensis* gastric mucosa identifies alterations in the AP-1 pathway induced by gastrin-mediated transformation. Physiol. Genomics 20: 131-142.
4. Yokoyama, A., et al. 2005. The Menin tumor suppressor protein is an essential oncogenic cofactor for MLL-associated leukemogenesis. Cell 123: 207-218.
5. Fontaniere, S., et al. 2006. Analysis of $p27^{Kip1}$ expression in Insulinomas developed in pancreatic β-cell specific Men1 mutant mice. Fam. Cancer 5: 49-54.
6. Hussein, N., et al. 2006. Reconstituted expression of Menin in Men1-deficient mouse Leydig tumour cells induces cell cycle arrest and apoptosis. Eur. J. Cancer 43: 402-414.
7. Lejonklou, M.H., et al. 2009. Neurogenin 3 and neurogenic differentiation 1 are retained in the cytoplasm of multiple endocrine neoplasia type 1 islet and pancreatic endocrine tumor cells. Pancreas 38: 259-266.
8. Lu, J., et al. 2010. α cell-specific Men1 ablation triggers the transdifferentiation of glucagon-expressing cells and Insulinoma development. Gastroenterology 138: 1954-1965.