McI-1 (MCL-2601): sc-69840



The Power to Question

BACKGROUND

B-cell CLL/lymphoma 2 (Bcl-2) blocks cell death following a variety of stimuli and confers a death-sparing effect to certain hematopoietic cell lines following growth factor withdrawal. Myeloid cell leukemia 1 (Mcl-1) shares sequence homology with Bcl-2 and further resembles Bcl-2 in that its expression promotes cell viability. p53 and Mcl-1 demonstrate opposing effects on mitochondrial apoptosis by mediating Bcl-2 antagonist killer (Bak) activity. Mcl-1 is an important and specific regulator that is necessary for the homeostasis of early hematopoietic progenitors. Glycogen synthase kinase 3 (GSK3) controls Mcl-1 stability, which has an effect on the regulation of apoptosis by growth factors, PI 3-kinase and Akt. Mice with a deficiency of the Mcl-1 protein show a significant reduction in B and T lymphocytes similar to the effects observed in IL-7- or IL-7R-deficient mice.

CHROMOSOMAL LOCATION

Genetic locus: MCL1 (human) mapping to 1q21.3; Mcl1 (mouse) mapping to 3 F2.1.

SOURCE

Mcl-1 (MCL-2601) is a mouse monoclonal antibody raised against recombinant full-length Mcl-1 of human origin.

PRODUCT

Each vial contains 200 μ g lgG₁ kappa light chain in 1.0 ml of PBS with < 0.1% sodium azide and 0.1% gelatin.

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.

APPLICATIONS

McI-1 (MCL-2601) is recommended for detection of McI-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), 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).

Suitable for use as control antibody for Mcl-1 siRNA (h): sc-35877, Mcl-1 siRNA (m): sc-35878, Mcl-1 shRNA Plasmid (h): sc-35877-SH, Mcl-1 shRNA Plasmid (m): sc-35878-SH, Mcl-1 shRNA (h) Lentiviral Particles: sc-35877-V and Mcl-1 shRNA (m) Lentiviral Particles: sc-35878-V.

Molecular Weight of McI-1 long form: 40 kDa.

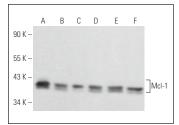
Molecular Weight of Mcl-1 short form: 32 kDa.

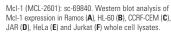
Positive Controls: Jurkat whole cell lysate: sc-2204, Ramos cell lysate: sc-2216 or HL-60 whole cell lysate: sc-2209.

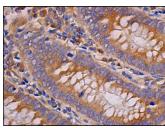
RECOMMENDED SUPPORT REAGENTS

To ensure optimal results, the following support reagents are recommended: 1) Western Blotting: use m-lgG κ BP-HRP: sc-516102 or m-lgG κ BP-HRP (Cruz Marker): sc-516102-CM (dilution range: 1:1000-1:10000), Cruz MarkerTM Molecular Weight Standards: sc-2035, UltraCruz® Blocking Reagent: sc-516214 and Western Blotting Luminol Reagent: sc-2048. 2) Immunoprecipitation: use Protein A/G PLUS-Agarose: sc-2003 (0.5 ml agarose/2.0 ml). 3) Immunofluorescence: use m-lgG κ BP-FITC: sc-516140 or m-lgG κ BP-PE: sc-516141 (dilution range: 1:50-1:200) with UltraCruz® Mounting Medium: sc-24941 or UltraCruz® Hard-set Mounting Medium: sc-359850. 4) Immunohistochemistry: use m-lgG κ BP-HRP: sc-516102 with DAB, 50X: sc-24982 and Immunohistomount: sc-45086, or Organo/Limonene Mount: sc-45087.

DATA







McI-1 (MCL-2601): sc-69840. Immunoperoxidase staining of formalin fixed, paraffin-embedded humar appendix tissue showing cytoplasmic staining of alandular cells.

SELECT PRODUCT CITATIONS

- Faiao-Flores, F., et al. 2013. Bcl-2 family proteins and cytoskeleton changes involved in DM-1 cytotoxic effect on melanoma cells. Tumour Biol. 34: 1235-1243.
- Tao, M., et al. 2022. Semaphorin 3F induces colorectal cancer cell chemosensitivity by promoting P27 nuclear export. Front. Oncol. 12: 899927.

PROTOCOLS

See our web site at www.scbt.com for detailed protocols and support products.



See McI-1 (22): sc-12756 for McI-1 antibody conjugates, including AC, HRP, FITC, PE, and Alexa Fluor® 488, 546, 594, 647, 680 and 790.

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