

Mcl-1 Rabbit monoclonal antibody

Catalog: BS9891M

Host: Rabbit

Reactivity: Human, Mouse, Rat

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 (GSK-3) 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.

Product:

Rabbit IgG, 1mg/ml in PBS with 0.02% sodium azide, 50% glycerol, pH7.2

Molecular Weight:

~ 29, 37 kDa

Swiss-Prot:

Q07820

Purification&Purity:

Protein A affinity purified

Applications:

WB: 1:1000

ICC/IF: 1:50-1:200

IHC: 1:50-1:200

FC: 1:50-1:100

Storage&Stability:

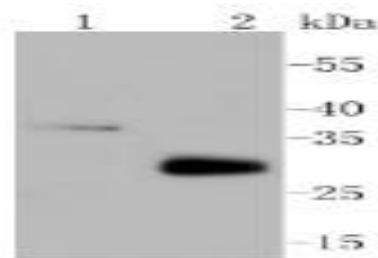
Store at 4 °C short term. Aliquot and store at -20 °C long term. Avoid freeze-thaw cycles.

Specificity:

This antibody detects endogenous levels of MCL1 and

does not cross-react with related proteins.

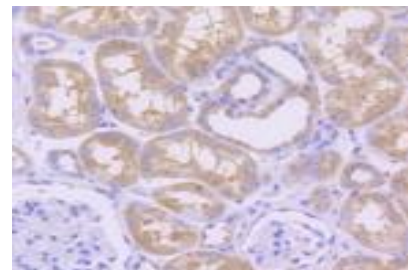
DATA:



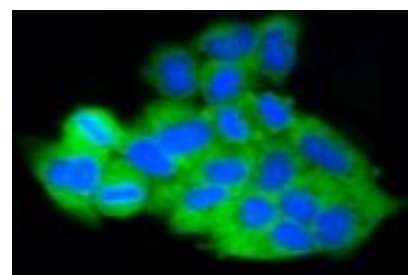
Western blot (WB) analysis of MCL1 Rabbit mAb at 1:1000 dilution

Lane1:Raji whole cell lysate

Lane2:The heart tissue lysate of Mouse



Immunohistochemical analysis of paraffin-embedded human kidney tissue using anti-MCL1 antibody. Counter stained with hematoxylin.



ICC staining MCL1 in HeLa cells (green). The nuclear counter stain is DAPI (blue). Cells were fixed in paraformaldehyde, permeabilised with 0.25% Triton *100/PBS.

Note:

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

Bioworld Technology, Inc.

Add: 1660 South Highway 100, Suite 500 St. Louis Park, MN 55416, USA.

Email: info@bioworld.com

Tel: 6123263284

Fax: 6122933841

Bioworld technology, co. Ltd.

Add: No 9, weidi road Qixia District Nanjing, 210046, P. R. China.

Email: info@biogot.com

Tel: 0086-025-68037686

Fax: 0086-025-68035151



PRODUCT DATA SHEET

Bioworld Technology, Inc.

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Add: 1660 South Highway 100, Suite 500 St. Louis Park,
MN 55416, USA.

Email: info@bioworld.com

Tel: 6123263284

Fax: 6122933841

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