

Mcl-1 (D2W9E) Rabbit mAb (PE Conjugate)

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For Research Use Only. Not for Use in Diagnostic Procedures.

Applications: FC-FP	Reactivity: H M R	Sensitivity: Endogenous	Source/Isotype: Rabbit IgG	UniProt ID: #P97287	Entrez-Gene Id: 17210
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Product Usage Information**Application**

Flow Cytometry (Fixed/Permeabilized)

Dilution

1:50

Storage

Supplied in PBS (pH 7.2), less than 0.1% sodium azide and 2 mg/ml BSA. Store at 4°C. Do not aliquot the antibody. Protect from light. Do not freeze.

Specificity/Sensitivity

Mcl-1 (D2W9E) Rabbit mAb (PE Conjugate) recognizes endogenous levels of total Mcl-1 protein.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Pro60 of mouse Mcl-1 protein.

Description

This Cell Signaling Technology antibody is conjugated to phycoerythrin (PE) and tested in-house for direct flow cytometry analysis in human cells. This antibody is expected to exhibit the same species cross-reactivity as the unconjugated Mcl-1 (D2W9E) Rabbit mAb #94296.

Background

Mcl-1 is an anti-apoptotic member of the Bcl-2 family originally isolated from the ML-1 human myeloid leukemia cell line during phorbol ester-induced differentiation along the monocyte/macrophage pathway (1). Similar to other Bcl-2 family members, Mcl-1 localizes to the mitochondria (2), interacts with and antagonizes pro-apoptotic Bcl-2 family members (3), and inhibits apoptosis induced by a number of cytotoxic stimuli (4). Mcl-1 differs from its other family members in its regulation at both the transcriptional and posttranslational level. First, Mcl-1 has an extended amino-terminal PEST region, which is responsible for its relatively short half-life (1,2). Second, unlike other family members, Mcl-1 is rapidly transcribed via a PI3K/Akt dependent pathway, resulting in its increased expression during myeloid differentiation and cytokine stimulation (1,5-7). Mcl-1 is phosphorylated in response to treatment with phorbol ester, microtubule-damaging agents, oxidative stress, and cytokine withdrawal (8-11). Phosphorylation at Thr163, the conserved MAP kinase/ERK site located within the PEST region, slows Mcl-1 protein turnover (10) but may prime the GSK-3 mediated phosphorylation at Ser159 that leads to Mcl-1 destabilization (11). Mcl-1 deficiency in mice results in peri-implantation lethality (12). In addition, conditional disruption of the corresponding *mcl-1* gene shows that Mcl-1 plays an important role in early lymphoid development and in the maintenance of mature lymphocytes (13).

Background References

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5. Wang, J.M. et al. (1999) *Mol Cell Biol* 19, 6195-206.
6. Jourdan, M. et al. (2003) *Oncogene* 22, 2950-9.
7. Chao, J.R. et al. (1998) *Mol Cell Biol* 18, 4883-98.
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11. Maurer, U. et al. (2006) *Mol Cell* 21, 749-60.
12. Rinckenberger, J.L. et al. (2000) *Genes Dev* 14, 23-7.
13. Opferman, J.T. et al. (2003) *Nature* 426, 671-6.

Species Reactivity

Species reactivity is determined by testing in at least one approved application (e.g., western blot).

Applications Key

FC-FP: Flow Cytometry (Fixed/Permeabilized)

Cross-Reactivity Key

H: Human **M:** Mouse **R:** Rat

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