Revision 1

Store at -20C

#14765

Phospho-Mcl-1 (Thr163) (D5M9D) Rabbit mAb

Cell Signaling TECHNOLOGY®

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

Applications:	Reactivity:	Sensitivity:	MW (kDa):	Source/Isotype:	UniProt ID:	Entrez-Gene Id:
W, IP	H	Endogenous	40	Rabbit IgG	#Q07820	4170

Product Usage Information

Application	Dilution
Western Blotting	1:1000
Immunoprecipitation	1:50

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Phospho-Mcl-1 (Thr163) (D5M9D) Rabbit mAb recognizes endogenous levels of Mcl-1 protein only when phosphorylated at Thr163. This antibody also cross-reacts with an unidentified protein of 70 kDa in some cell lines.

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Thr163 of human Mcl-1 protein.

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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3. Sato, T. et al. (1994) *Proc Natl Acad Sci USA* 91, 9238-42.
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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. Rinkenberger, 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).

Western Blot Buffer

IMPORTANT: For western blots, incubate membrane with diluted primary antibody in 5% w/v BSA, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

Applications Key

W: Western Blotting **IP:** Immunoprecipitation

Cross-Reactivity Key

H: Human

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