

# Phospho-Mcl-1 (Ser64) Antibody



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<b>Applications:</b> W	<b>Reactivity:</b> H	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 40	<b>Source/Isotype:</b> Rabbit	<b>UniProt ID:</b> #Q07820	<b>Entrez-Gene Id:</b> 4170
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## Product Usage Information

### Application

Western Blotting

### Dilution

1:1000

## Storage

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

## Specificity/Sensitivity

Phospho-Mcl-1 (Ser64) Antibody recognizes endogenous levels of Mcl-1 protein only when phosphorylated at Ser64. Non-specific bands of unknown origin are detected in some cell lines at 70 and 140 kDa.

## Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic phosphopeptide corresponding to residues surrounding Ser64 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). Phosphorylation of Mcl-1 at Ser64 can target it for ubiquitination and destruction by the tumor suppressor protein FBW7 (14,15)

## Background References

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3. Sato, T. et al. (1994) *Proc Natl Acad Sci USA* 91, 9238-42.
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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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12. Rinckenberger, J.L. et al. (2000) *Genes Dev* 14, 23-7.
13. Opferman, J.T. et al. (2003) *Nature* 426, 671-6.
14. Inuzuka, H. et al. (2011) *Nature* 471, 104-9.
15. Wertz, I.E. et al. (2011) *Nature* 471, 110-4.

## 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 nonfat dry milk, 1X TBS, 0.1% Tween® 20 at 4°C with gentle shaking, overnight.

## Applications Key

**W:** Western Blotting

**Cross-Reactivity Key****H:** Human**Trademarks and Patents**

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