

Menin (D45B1) XP[®] Rabbit mAb

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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, IF-IC	H M R Mk	Endogenous	76	Rabbit IgG	#O00255	4221

Product Usage Information

Application

Western Blotting
Immunofluorescence (Immunocytochemistry)

Dilution

1:1000
1:50 - 1:200

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

Menin (D45B1) XP[®] Rabbit mAb recognizes total endogenous levels of all 3 isoforms of Menin protein.

Species predicted to react based on 100% sequence homology

Bovine, Pig, Horse

Source / Purification

Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to residues surrounding Val597 of human Menin protein.

Background

Mutations in the *MEN1* tumor suppressor gene cause multiple endocrine neoplasia type 1 (MEN1), an autosomal dominant familial tumor syndrome typified by tumors of the pituitary, parathyroid, lung, and enteropancreatic endocrine tissues (1,2). Patients with this tumor syndrome have inherited either missense or truncation mutations in one allele of the *MEN1* gene, while the other allele is subject to loss of heterozygosity in tumors from these patients (1,2). Menin, the protein product of the *MEN1* gene, is a component of the mixed-lineage leukemia protein (MLL)-containing histone methyltransferase complex that facilitates methylation of histone H3 Lys4 to promote transcriptional activation (3,4). Menin functions to suppress proliferation of pancreatic islet cells, at least in part through MLL-mediated activation of the *p18* and *p27* cyclin-dependent kinase inhibitor genes (5,6). Loss of Menin leads to a decrease in methylation of histone H3 Lys4 and decreased expression of the *p18* and *p27* genes, leading to hyperplasia (5,6). In contrast to its role as a tumor suppressor in endocrine cells, Menin has been shown to promote proliferation in leukemia cells driven by MLL-fusion proteins. Menin is essential for oncogenic MLL-fusion-protein-mediated transformation of bone marrow cells and is required for histone H3 Lys4 methylation and expression of the *HoxA9* gene (7,8). Menin interacts with a wide range of proteins, including JunD, SMAD family members, estrogen receptor, vitamin D receptor, PEM, NFκB, FANCD2, RPA2, NMMHC II-A, GFAP, vimentin, and HSP70, suggesting additional roles in transcriptional regulation, DNA processing and repair, cytoskeleton organization, and protein degradation (9,10).

Background References

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- Lemmens, I. et al. (1997) *Hum Mol Genet* 6, 1177-83.
- Hughes, C.M. et al. (2004) *Mol Cell* 13, 587-97.
- Yokoyama, A. et al. (2004) *Mol Cell Biol* 24, 5639-49.
- Karnik, S.K. et al. (2005) *Proc Natl Acad Sci USA* 102, 14659-64.
- Schnepp, R.W. et al. (2006) *Cancer Res* 66, 5707-15.
- Yokoyama, A. et al. (2005) *Cell* 123, 207-18.
- Chen, Y.X. et al. (2006) *Proc Natl Acad Sci USA* 103, 1018-23.
- Agarwal, S.K. et al. (2005) *Horm Metab Res* 37, 369-74.
- Wu, X. and Hua, X. (2008) *Curr Mol Med* 8, 805-15.

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 **IF-IC:** Immunofluorescence (Immunocytochemistry)

Cross-Reactivity Key

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

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