

## **ZMYND8 Antibody**



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

<b>Applications:</b> W, IP	Reactivity: H Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 180	Source/Isotype: Rabbit	<b>UniProt ID:</b> #Q9ULU4	Entrez-Gene Id: 23613
Product Usage Information	•	<b>Application</b> Western Blotting Immunoprecipitation			<b>Dilution</b> 1:1000 1:50	
Storage		Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 $\mu$ g/ml BSA and 50% glycerol. Store at – 20°C. Do not aliquot the antibody.				
Specificity/Sensitivity		ZMYND8 Antibody recognizes endogenous levels of total ZMYND8 protein.				
Source / Purification		Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to residues near the amino terminus of human ZMYND8 protein. Antibodies are purified by protein A and peptide affinity chromatography.				
Background		Zinc finger MYND domain-containing protein 8 (ZMYND8), also referred to as receptor for activated C-kinase 7 (Rack7) and protein kinase C-binding protein 1 (PRKCBP1), is a DNA damage response protein and a transcriptional regulator that is a close homolog of ZMYND11 (BS69) (1). ZMYND8 binds to H3K36me2 and H4K16ac, two histone marks associated with active transcription (2). This protein is targeted to sites of DNA damage within actively transcribed genes, and recruits the H3K4me3-specific histone demethylase KDM5A/JARID1A and nucleosome remodeling and histone deacetylation (NuRD) complex (1-3). Together, these protein complexes mediate transcriptional repression and allow for subsequent double-strand break repair via homologous recombination. ZMYND8 contains a bromodomain and a PWWP domain near its N-terminus, and a MYND domain towards the C-terminus, the latter of which mediates interaction with the NuRD complex (1). ZMYND8 also functions to recruit the H3K4me3-specific histone demethylase KDM5C/JARID1C to enhancer and super-enhancer regions, and functions as a negative regulator of gene expression (4). ZMYND8 and JARID1C are both putative tumor suppressor proteins, and knockdown of either of these proteins leads to derepression of S100 oncogenes (1). ZMYND8 expression is altered in breast and cervical cancer (4,5), and has been found to be translocated with RELA in at least one patient with acute erythroid leukemia (6). Knockdown of ZMYND8 expression in breast cancer cell lines increases anchorage-independent cell growth, cell migration and invasion, and tumor growth in mouse xenograft models (4).				
Background References		<ol> <li>Gong, F. et al. (2015) <i>Genes Dev</i> 29, 197-211.</li> <li>Adhikary, S. et al. (2016) <i>J Biol Chem</i> 291, 2664-81.</li> <li>Gong, F. et al. (2017) <i>J Cell Biol</i> 216, 1959-74.</li> <li>Shen, H. et al. (2016) <i>Cell</i> 165, 331-42.</li> <li>Bierkens, M. et al. (2013) <i>Genes Chromosomes Cancer</i> 52, 56-68.</li> <li>Panagopoulos, I. et al. (2013) <i>PLoS One</i> 8, e63663.</li> </ol>				

**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 Mk: Monkey

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