## SET7/SET9 (C24B1) Rabbit mAb



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

Applications: W	Reactivity: H M R Mk	<b>Sensitivity:</b> Endogenous	<b>MW (kDa):</b> 48	<b>Source/Isotype:</b> Rabbit IgG	UniProt ID: #Q8WTS6	Entrez-Gene Id: 80854
Product Usage Information		<b>Application</b> Western Blotting			<b>Dilution</b> 1:1000	
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		SET7/SET9 (C24B1) Rabbit mAb detects endogenous levels of total SET7/SET9 protein. This antibody does not cross-react with other SET domain-containing proteins.				
Source / Purification		Monoclonal antibody is produced by immunizing animals with a synthetic peptide corresponding to the amino terminus of the human SET7/SET9 protein.				
Background		SET7/SET9 is a member of the SET domain-containing family, and can specifically methylate Lys4 on histone H3 (1). Like most other lysine-directed histone methyltransferases, it contains a conserved catalytic SET domain originally identified in the Drosophila Su(var)3-9, Enhancer of zeste and Trithorax proteins. Histone methylation is a major determinant for the formation of active and inactive regions of the genome and is crucial for the proper programming of the genome during development (2,3). Methylation of histone H3 Lys4 enhances transcriptional activation by coordinating the recruitment of BPTF, a component of the NURF chromatin remodeling complex, and WDR5, a component of multiple histone methyltransferase complexes (4,5). In addition, methylation of lysine 4 blocks transcriptional repression by inhibiting the binding of the NURD histone deacetylation complex to the amino-terminal tail of histone H3 and interfering with SUV39H1-mediated methylation of histone H3 Lys9 (1). SET7/SET9 is highly active on free histone H3, but only very weakly methylates H3 within nucleosomes (1). Besides histones, SET7/SET9 also methylates Lys189 of the TAF10, a member of the TFIID transcription factor complex, and Lys372 of the p53 tumor suppressor protein (6,7). Methylation of TAF10 stimulates transcription in a promoter-specific manner by increasing the affinity of TAF10 for RNA polymerase II, which may potentiate pre-initiation complex formation (6). Methylation of p53 at Lys372 increases protein stability and leads to upregulation of target genes such as p21. Thus the loss of SET7/SET9 may represent another mechanism for the inactivation of p53 in human cancers (7).				
Background References		1. Nishioka, K. et al. (2002) <i>Genes Dev.</i> 16, 479-489. 2. Kubicek, S. et al. (2006) <i>Ernst Schering Res. Found. Workshop</i> , 1-27. 3. Lin, W. and Dent, S.Y. (2006) <i>Curr. Opin. Genet. Dev.</i> 16, 137-142. 4. Wysocka, J. et al. (2006) <i>Nature</i> 442, 86-90. 5. Wysocka, J. et al. (2005) <i>Cell</i> 121, 859-872. 6. Kouskouti, A. et al. (2004) <i>Mol. Cell</i> 14, 175-182. 7. Chuikov, S. et al. (2004) <i>Nature</i> 432, 353-360.				
Species Reactiv	rity	Species reactivity is d	etermined by testin	g in at least one approve	ed 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

**Cross-Reactivity Key** H: Human M: Mouse R: Rat Mk: Monkey

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