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Recombinant SET7 Monoclonal Antibody

catalog number: AN302013L

Note: Centrifuge before opening to ensure complete recovery of vial contents.

Description

Reactivity Human; Rat; Mouse

Immunogen Peptide. This information is proprietary to PTMab.

HostRabbitIsotypeIgG, κCloneA733

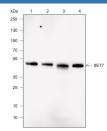
Purification Protein A purified

Buffer PBS, 50% glycerol, 0.05% Proclin 300, 0.05% protein protectant.

Applications Recommended Dilution

WB 1:1000

Data



Western Blot with SET7 Monoclonal Antibody at dilution of

1:1000. Lane 1: HeLa, Lane 2: Jurkat, Lane 3: NIH/3T3,

Lane 4: PC-12

Observed-MW:47 kDa Calculated-MW:41 kDa

Preparation & Storage

Storage Storage Store at -20°C Valid for 12 months. Avoid freeze / thaw cycles.

Shipping Ice bag

Background

For Research Use Only

 Toll-free: 1-888-852-8623
 Tel: 1-832-243-6086
 Fax: 1-832-243-6017

 Web: www.elabscience.com
 Email: techsupport@elabscience.com
 Rev. V1.0

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SET7/SET9 is a member of the SET domain-containing family, and can specifically methylate Lys4 on histone H3. 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. 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. 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. SET7/SET9 is highly active on free histone H3, but only very weakly methylates H3 within nucleosomes. 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. 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. 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.

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