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SUV39H1 Antibody (N-term) Blocking peptide

Synthetic peptide
Catalog # BP14022a

Specification

SUV39H1 Antibody (N-term) Blocking peptide - Product Information

Primary Accession O43463

SUV39H1 Antibody (N-term) Blocking peptide - Additional Information

Gene ID 6839

Other Names
Histone-lysine N-methyltransferase SUV39H1, Histone H3-K9 methyltransferase 1, H3-K9-HMTase 1, Lysine N-methyltransferase 1A, Position-effect variegation 3-9 homolog, Suppressor of variegation 3-9 homolog 1, Su(var)3-9 homolog 1, SUV39H1, KMT1A, SUV39H

Target/Specificity
The synthetic peptide sequence used to generate the antibody AP14022a was selected from the N-term region of SUV39H1. A 10 to 100 fold molar excess to antibody is recommended. Precise conditions should be optimized for a particular assay.

Format
Peptides are lyophilized in a solid powder format. Peptides can be reconstituted in solution using the appropriate buffer as needed.

Storage
Maintain refrigerated at 2-8°C for up to 6 months. For long term storage store at -20°C.

Precautions
This product is for research use only. Not for use in diagnostic or therapeutic procedures.

SUV39H1 Antibody (N-term) Blocking peptide - Protein Information

Name SUV39H1

Synonyms KMT1A, SUV39H

Function
Histone methyltransferase that specifically trimethylates 'Lys-9' of histone H3 using monomethylated H3 'Lys-9' as substrate. Also weakly methylates histone H1 (in vitro). H3 'Lys-9' trimethylation represents a specific tag for epigenetic transcriptional repression by recruiting HP1 (CBX1, CBX3 and/or CBX5) proteins to methylated histones. Mainly functions in heterochromatin regions, thereby playing a central role in the establishment of constitutive heterochromatin at pericentric and telomere regions. H3 'Lys-9' trimethylation is also required to direct DNA methylation at pericentric repeats. SUV39H1 is targeted to histone H3 via its interaction with RB1 and is involved in many processes, such as repression of MYOD1-stimulated differentiation, regulation of the control switch for exiting the cell cycle and entering differentiation, repression by

the PML-RARA fusion protein, BMP-induced repression, repression of switch recombination to IgA and regulation of telomere length. Component of the eNoSC (energy-dependent nucleolar silencing) complex, a complex that mediates silencing of rDNA in response to intracellular energy status and acts by recruiting histone-modifying enzymes. The eNoSC complex is able to sense the energy status of cell: upon glucose starvation, elevation of NAD(+)/NADP(+) ratio activates SIRT1, leading to histone H3 deacetylation followed by dimethylation of H3 at 'Lys-9' (H3K9me2) by SUV39H1 and the formation of silent chromatin in the rDNA locus. Recruited by the large PER complex to the E-box elements of the circadian target genes such as PER2 itself or PER1, contributes to the conversion of local chromatin to a heterochromatin-like repressive state through H3 'Lys-9' trimethylation.

Cellular Location
Nucleus. Nucleus lamina. Nucleus, nucleoplasm Chromosome, centromere. Note=Associates with centromeric constitutive heterochromatin

SUV39H1 Antibody (N-term) Blocking peptide - Protocols

Provided below are standard protocols that you may find useful for product applications.

- Blocking Peptides

SUV39H1 Antibody (N-term) Blocking peptide - Images

SUV39H1 Antibody (N-term) Blocking peptide - Background

This gene is a member of the suppressor of variegation 3-9homolog family and encodes a protein with a chromodomain and aC-terminal SET domain. This nuclear protein moves to thecentromeres during mitosis and functions as a histonemethyltransferase, methylating Lys-9 of histone H3. Overall, itplays a vital role in heterochromatin organization, chromosomesegregation, and mitotic progression.

SUV39H1 Antibody (N-term) Blocking peptide - References

Chen, L., et al. EMBO J. 29(15):2538-2552(2010)Goyama, S., et al. Leukemia 24(1):81-88(2010)Cherrier, T., et al. Oncogene 28(38):3380-3389(2009)Li, Z., et al. J. Biol. Chem. 284(16):10361-10366(2009)Spensberger, D., et al. FEBS Lett. 582(18):2761-2767(2008)