Revision 1

Store at -20C

#26218

Histone H3 (K36M Mutant Specific) Antibody

Cell Signaling TECHNOLOGY®

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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	H M	Endogenous	17	Rabbit	#P84243	3020

Product Usage Information

Application	Dilution
Western Blotting	1:1000

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA and 50% glycerol. Store at –20°C. Do not aliquot the antibody.

Specificity/Sensitivity

Histone H3 (K36M Mutant Specific) Antibody recognizes endogenous levels of K36M mutant histone H3.1, H3.2, and H3.3 proteins. The antibody may show slight cross-reactivity with wild-type histone H3.1, 3.2, or 3.3 when used at a high concentration. Careful titration of this antibody may be required to obtain optimal specificity.

Species predicted to react based on 100% sequence homology

Rat

Source / Purification

Polyclonal antibodies are produced by immunizing animals with a synthetic peptide corresponding to K36M mutant sequence of human histone H3.3 protein. Antibodies are purified by protein A and peptide affinity chromatography.

Background

Chondroblastoma is a rare type of benign tumor that is found at the rounded ends of the long bones in the arms and legs. More than 90% of chondroblastomas have been found to contain a heterozygous mutation in the *H3F3A* gene encoding the histone variant H3.3 (1). This mutation, a lysine to methionine amino acid substitution in codon 36 (K36M), inhibits at least two histone H3 lysine 36 methyltransferases, WHSC1 (MMSET) and SETD2, resulting in reduced global levels of histone H3 lysine 36 methylation (1). Chondrocytes containing the histone H3 K36M mutation exhibit several hallmarks of cancer cells, including increased ability to form colonies, resistance to apoptosis, and defects in differentiation. Reduction of global methylation levels in chondrocytes, resulting from the K36M mutation, contributes to tumorigenesis by altering the expression of cancer-associated genes. The histone H3 K36M mutation is also found to promote sarcomagenesis by impairing the differentiation of mesenchymal progenitor cells, resulting in undifferentiated sarcomas (2). The K36M mutation alters the histone methylation landscape, resulting in a genome-wide gain in histone H3 lysine 27 methylation, redistribution of polycomb repressive complex 1, and derepression of its target genes known to block mesenchymal differentiation. Finally, the histone H3 K36M mutation is also found in 13% of HPV-negative head and neck squamous cell carcinomas, again contributing to tumorigenesis by altering global methylation levels of histone H3 lysine 36 (3).

Background References

1. Fang, D. et al. (2016) *Science* 352, 1344-8.
2. Lu, C. et al. (2016) *Science* 352, 844-9.
3. Papillon-Cavanagh, S. et al. (2017) *Nat Genet* 49, 180-5.

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

Cross-Reactivity Key

H: Human **M:** Mouse

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