

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide
Synthetic peptide
Catalog # BP1197c**Specification**

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Product InformationPrimary Accession [Q96KQ7](#)**KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Additional Information****Gene ID** 10919**Other Names**

Histone-lysine N-methyltransferase EHMT2, 211-, Euchromatic histone-lysine N-methyltransferase 2, HLA-B-associated transcript 8, Histone H3-K9 methyltransferase 3, H3-K9-HMTase 3, Lysine N-methyltransferase 1C, Protein G9a, EHMT2, BAT8, C6orf30, G9A, KMT1C, NG36

Target/Specificity

The synthetic peptide sequence used to generate the antibody [AP1197c](/product/products/AP1197c) was selected from the Center region of human G9a. 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.

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Protein Information**Name** EHMT2**Synonyms** BAT8, C6orf30, G9A, KMT1C, NG36**Function**

Histone methyltransferase that specifically mono- and dimethylates 'Lys-9' of histone H3 (H3K9me1 and H3K9me2, respectively) in euchromatin. H3K9me represents a specific tag for epigenetic transcriptional repression by recruiting HP1 proteins to methylated histones. Also mediates monomethylation of 'Lys-56' of histone H3 (H3K56me1) in G1 phase, leading to promote interaction between histone H3 and PCNA and regulating DNA replication. Also weakly methylates 'Lys-27' of histone H3 (H3K27me). Also required for DNA methylation, the histone methyltransferase activity is not required for DNA methylation, suggesting that these 2 activities function independently. Probably targeted to histone H3 by different DNA-binding proteins like

E2F6, MGA, MAX and/or DP1. May also methylate histone H1. In addition to the histone methyltransferase activity, also methylates non-histone proteins: mediates dimethylation of 'Lys-373' of p53/TP53. Also methylates CDYL, WIZ, ACIN1, DNMT1, HDAC1, ERCC6, KLF12 and itself. Recruited to the promoters of target genes through interaction with transcriptional repressor MSX1, leading to the inhibition of myoblast differentiation via transcriptional repression of differentiation factors (By similarity).

Cellular Location

Nucleus. Chromosome. Note=Associates with euchromatic regions (PubMed:11316813). Does not associate with heterochromatin (PubMed:11316813).

Tissue Location

Expressed in all tissues examined, with high levels in fetal liver, thymus, lymph node, spleen and peripheral blood leukocytes and lower level in bone marrow

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Protocols

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

- [Blocking Peptides](#)

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Images**KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - Background**

A cluster of genes, BAT1-BAT5, has been localized in the vicinity of the genes for TNF alpha and TNF beta. The gene for G9a is found near this cluster; it was mapped near the gene for C2 within a 120-kb region that included a HSP70 gene pair. These genes are all within the human major histocompatibility complex class III region. The protein encoded by this gene is thought to be involved in intracellular protein-protein interaction. There are three alternatively spliced transcript variants of this gene but only two are fully described.

KMT1C / G9a / BAT8 Antibody (Center) Blocking peptide - References

Tachibana, M., et al., Genes Dev. 16(14):1779-1791 (2002).Brown, S.E., et al., Mamm. Genome 12(12):916-924 (2001).Spies, T., et al., Proc. Natl. Acad. Sci. U.S.A. 86(22):8955-8958 (1989).Milner, C.M., et al., Biochem. J. 290 (Pt 3), 811-818 (1993).