

**KMT1C Antibody (C-term) Blocking peptide**  
**Synthetic peptide**  
**Catalog # BP1197b****Specification**

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**KMT1C Antibody (C-term) Blocking peptide - Product Information**Primary Accession [Q96KQ7](#)**KMT1C Antibody (C-term) 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 [AP1197b](/product/products/AP1197b) was selected from the C-term region of human BAT8. 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 Antibody (C-term) 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.

**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 Antibody (C-term) Blocking peptide - Protocols**

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

- [Blocking Peptides](#)

**KMT1C Antibody (C-term) Blocking peptide - Images****KMT1C Antibody (C-term) Blocking peptide - Background**

BAT8, also known as G9A, is a lysine-preferring histone methyltransferase. H3 Lys-9 methylation represents a specific tag for epigenetic transcriptional repression by recruiting HP1 proteins to methylated histones. The presence of the ankyrin repeats also suggests that BAT8 is involved in intracellular protein-protein interaction. Like Suv39h1, BAT8 transfers methyl groups to the lysine residues of histone H3, but with a 10- to 20-fold higher activity. BAT8 was able to add methyl groups to lys27 as well as lys9 in histone H3, compared with Suv39h1, which was able only to methylate lys9. BAT8 is localized to the nucleus but not in the repressive chromatin domains of centromeric loci, in which Suv39h1 family proteins were localized. This protein is probably targeted to histone H3 by different DNA-binding proteins like E2F6, MGA, MAX and/or DP1. BAT8 also methylates histone H1.

**KMT1C Antibody (C-term) 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).