

#97763 Store at -20°C

DNA Cytosine Modification Antibody Sampler Kit

1 Kit (4 x 20 microliters)

**Cell Signaling**
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For Research Use Only. Not for Use in Diagnostic Procedures.

Product Includes	Product #	Quantity	Mol. Wt	Isotype/Source
5-Methylcytosine (5-mC) (D3S2Z) Rabbit mAb	28692	20 µl		Rabbit IgG
5-Hydroxymethylcytosine (5-hmC) (HMC31) Mouse mAb	51660	20 µl		Mouse IgG1
5-Carboxylcytosine (5-caC) (D7S8U) Rabbit mAb	36836	20 µl		Rabbit IgG
5-Formylcytosine (5-fC) (D5D4K) Rabbit mAb	74178	20 µl		Rabbit IgG
Anti-rabbit IgG, HRP-linked Antibody	7074	100 µl		Goat
Anti-mouse IgG, HRP-linked Antibody	7076	100 µl		Horse

Please visit cellsignal.com for individual component applications, species cross-reactivity, dilutions, protocols, and additional product information.

Description

The DNA Cytosine Modification Antibody Sampler Kit provides an economical means of detecting the levels of cytosine modifications in DNA by dot blot using antibodies against 5-methylcytosine, 5-hydroxymethylcytosine, 5-formylcytosine, and 5-carboxylcytosine.

Storage

Supplied in 10 mM sodium HEPES (pH 7.5), 150 mM NaCl, 100 µg/ml BSA, 50% glycerol and less than 0.02% sodium azide. Store at -20°C. Do not aliquot the antibody.

Background

Methylation of DNA at cytosine residues is a heritable, epigenetic modification that is critical for proper regulation of gene expression, genomic imprinting, and mammalian development (1,2). 5-methylcytosine is a repressive epigenetic mark established *de novo* by two enzymes, DNMT3a and DNMT3b, and is maintained by DNMT1 (3, 4). 5-methylcytosine was originally thought to be passively depleted during DNA replication. However, subsequent studies have shown that Ten-Eleven Translocation (TET) proteins TET1, TET2, and TET3 can catalyze the oxidation of methylated cytosine to 5-hydroxymethylcytosine (5-hmC) (5). Additionally, TET proteins can further oxidize 5-hmC to form 5-formylcytosine (5-fC) and 5-carboxylcytosine (5-caC), both of which are excised by thymine-DNA glycosylase (TDG), effectively linking cytosine oxidation to the base excision repair pathway and supporting active cytosine demethylation (6,7). TET protein-mediated cytosine hydroxymethylation was initially demonstrated in mouse brain and embryonic stem cells (5, 8). Since then this modification has been discovered in many tissues, with the highest levels found in the brain (9). While 5-fC and 5-caC appear to be short-lived intermediate species, there is mounting evidence showing that 5-hmC is a distinct epigenetic mark with various unique functions (10,11). The modified base itself is stable *in vivo* and interacts with various readers, including MeCP2 (11,12). The global level of 5-hmC increases during brain development and 5-hmC is enriched at promoter regions and poised enhancers. Furthermore, there is an inverse correlation between levels of 5-hmC and histone H3K9 and H3K27 trimethylation, suggesting a role for 5-hmC in gene activation (12). Lower amounts of 5-hmC have been reported in various cancers, including myeloid leukemia and melanoma (13,14).

**Background
References**

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