Epigenetics antibodies

Covering 90% of epigenetic targets and transcription factors

Epigenetics

Epigenetics is the study of heritable changes in gene expression that modify DNA, RNA, and protein but do not alter the nucleotide sequence. Posttranslational modifications (PTMs) are among the most important types of epigenetic states that apply to proteins. PTMs are marks that provide an extensive regulatory mechanism for cells to signal which genes to turn on and off. Many types and families of proteins are subject to PTMs, but one of the most highly decorated is the histone family of proteins. Some examples of PTMs are methylation, acetylation, phosphorylation, and ubiquitination.

It is essential to use an antibody specific to an individual histone modification because each one represents a unique signal for gene expression. For example, Lys9 on H3 can be acetylated or methylated. Acetylation is an activating mark, whereas methylation has different effects depending on the number of methyl groups. H3K9me1 is found to be enriched at transcription start sites, whereas H3K9me2 and H3K9me3 are associated with gene repression. Further, H3K9me2 is specifically associated with X-chromosome inactivation. Thus, each modification on H3K9 has a distinct effect on the cell, and knowing the identity of the modification is essential for accurately characterizing expression (Figure 1).

Graphical representation of selectivity of H3K9me2 antibody

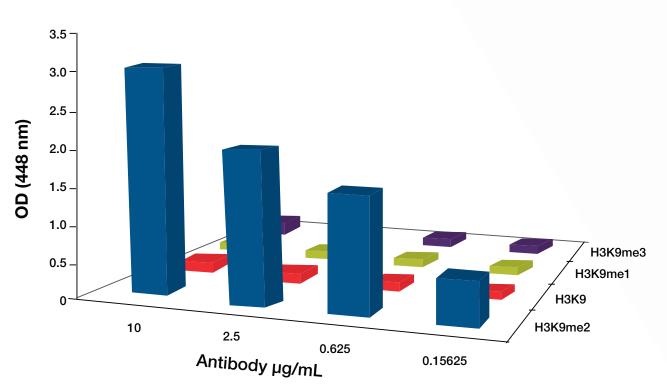
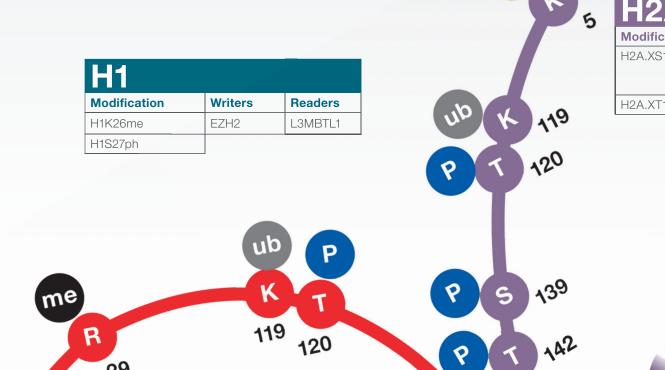


Figure 1. Cross-reactivity in ELISA for Invitrogen[™] ABfinity[™] anti-H3K9me2 rabbit recombinant monoclonal antibody (Cat. No. 701783).



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11	

Modification H2AS1ph H2AR3me	Writers MSK1, PKC	me
· ·	MSK1, PKC	
H2AR3ma	I .	
112/41101110	PRMT1, PRMT6	
H2AK5ac	TIP60, p300, CBP, KAT1, KAT5	
H2AR11me	PRMT1, PRMT6	
H2AR29me	PRMT1, PRMT6	
H2AK119ub	Ring2, Ring1A	ac
H2AT120ph	Bub1, VprBP, NHK-1	
		P

			6	
	we	Pme	45	
		K 41	42	
m 20 5	09	36		
me R 21	28	m	e	
26 23		SC B	T P 2	
ac		We K	3	
18 R 17	me me	PT	n	
me K 11 P		R		
14	5 10 9	8		

Modification	Writers	Readers	Erasers
H3R2me	PRMT4, PRMT6		
H3T3ph	GSG2, VRK1	Survivin	
H3K4ac			SIRT1, SIRT2, SIRT3, HDAC1, HDAC2, HDAC3
H3K4me	MLL1, MLL2, MLL3, MLL4, MLL5, SETD1A, SETD1B, ASCL1, SETD7, NSD3	CHD1, MRG15, PHF20L1, TAF3, ING1, ING2, ING3, ING4, ING5, BPTF, RAG2, ATRX, PHF8, KDM4A, KDM4C, ZCWPW1	LSD1, LSD2, KDM2B, JARID1A, JARID1B, JARID1C, JARID1D, PHF8, NO66
H3T6ph	PKC-beta		
H3R8me	PRMT5		
H3K9ac	GCN5, PCAF, ELP3	BRD4, BAZ1B	SIRT6, SIRT1
H3K9me	SUV39H1, SUV39H2, G9a, SETDB1, ASCL1, KMT1D, CLL8, RIZ1	L3MBTL1, TIP60, SFMBT, HP1, CDY1, PC1, MPP8, CBX1, CBX2, CBX3, CBX4, CBX5, CBX6, CBX7, CBX8, Np95, JARID1C, ATRX, SMCX, TDRD7, EED, G9a	LSD1, KMD3A, KMD3B, KMD4A, KMD4B, KMD4C, KMD4D, TRIP8, PHF8
H3S10ph	Aurora-B, MSK1, IKK-alpha, SIK1, MSK2, PIM1	14-3-3	PPF
H3T11ph	ZIPK		
H3K14ac	GCN5, PCAF, CBP, p300, TIP60, SRC-1, ELP3, KAT12, TAF1, MOZ, MORF	BRD4, BAZ1B, BRG1, DPF3b	
H3R17me	PRMT4	TDRD3	
H3K18ac	GCN5, p300, CBP, PCAF, KAT12		
H3K23ac	GCN5, MYST2, p300, CBP, KAT3A, KAT3B		
H3R26me	PRMT4		
H3K27ac	GCN5, p300, CBP		
H3K27me	EZH2, G9a, EZH1, NSD3	PC1, CBX2, CBX4, CBX6, CBX7, CBX8, EED, CDY, MPP8	UTX, JMJD3, PHF8
H3S28ph	Aurora-B, MSK1, MSK2	14-3-3	
H3K36ac	GCN5, PCAF		
H3K36me	SETD2, NSD1, SMYD2, NSD2, ASCL1, SETMAR	MSL3, MRG15, BRPF1, PHF19, PHF1, DNMT3A, NSD1, NSD2, NSD3	KDM2A, KDM2B, KDM4A, KDM4B, KDM4C, NO66
H3Y41ph	JAK2		
H3R42me	CARM1		
H3Y45ph	PKC-delta		
H3K56ac	GCN5, CBP, p300		HDAC1, HDAC2, SIRT2, SIRT6
H3K79me	KMT4	53BP1	

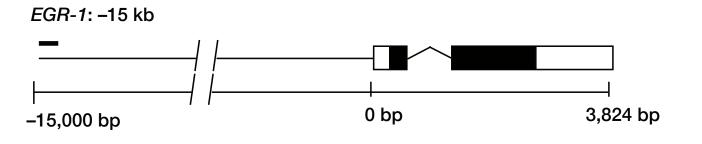
H2B	I
Modification	Writers
H2BK5ac	p300, ATF2
H2BK12ac	p300, CBP, ATF2
H2BS14ph	MST1
H2BK15ac	p300, CBP, ATF2
H2BK20ac	p300
H2BS33ph	TAF1
H2BS36ph	AMPK
H2BK120ub	UBE2E1, RNF20, RNF40,

Modification	Writers	Readers	Erasers
H4S1ph	CKII		
H4R3me	PRMT 1, PRMT5, PRMT6	TDRD3, PCAF, p300	JMJD6
H4K5ac	HAT1, TIP60, ATF2, p300, CBP, HBO1	BRD4	
H4K8ac	GCN5, TIP60, ATF2, ELP3, p300, CBP, HBO1	BRD2, BRD4	
H4K12ac	HAT1, TIP60, p300, CBP	BRD2, BRD4	
H4K16ac	GCN5, MOF, TIP60, ATF2	GCN5	SIRT2, SIRT1
H4K20me	PR-SET7, SUV4-20H1, SUV20-H2, ASCL1, NSD1, SETD8	PHF20L1, L3MBTL1, SFMBT, MBTD1, 53BP1, LRWD1	
H4K91ac	HAT4, GCN5		
H4K91ub	DTXL3		

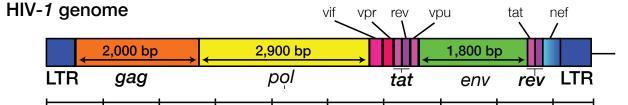
Writers, readers, and erasers

Epigenetic regulation is a dynamic process and includes writers, readers, and erasers. Writers place a PTM mark on a specific amino acid on histones or other proteins. These include histone acetyltransferases (HATs), histone methyltransferases (HMTs), protein arginine methyltransferases (PRMTs), and kinases. Readers bind to the epigenetic marks and include proteins with bromodomains, chromodomains, and tudor domains. Epigenetic erasers remove such marks and include histone deacetylases (HDACs), lysine demethylases (KDMs), and phosphatases. The writing, reading, and erasing of these posttranslational marks lead to changes in chromatin structure that can promote or antagonize gene expression. The identification of writers, readers, and erasers is continually growing.

EGR-1: -15 kb
Forward: GAGGCACTCTGCTCACCAAA
Reverse: GATGCCTGCGAGGATGGAAA



HIV gag
Forward: GCTGAGCTGAGCTTCGGTTC
Reverse: TCGCCGCCTACTCAGTAGGTA



0 1,000 2,000 3,000 4,000 5,000 6,000 7,000 8,000 9,000 9,719

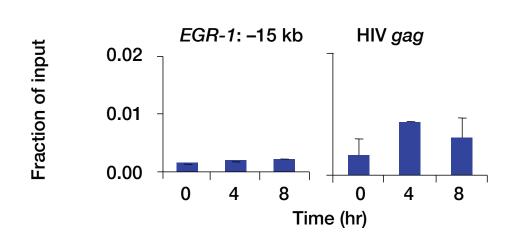


Figure 2. Matrix ChIP analysis using Invitrogen™ GCN5 Antibody (Cat. No. MA3-046) performed on a culture of human 5A8 J-lat T lymphocytes latently infected with HIV-1 and treated with 10 μg/mL phytohemagglutinin (PHA) for the indicated times.









