PTM BIO
Pioneer in Proteomics

15 Years of Innovation in PTM Antibodies

First to create antibodies for novel PTMs including lactylation, crotonylation, malonylation, and more.

Explore by Modification Types

Phosphorylation (P)
Discovered in the 1950s

Serotonylation (Ser)
Discovered in the 1950s

Citrullination (Cit)
Discovered in the 1950s

Methylation (Me)
Discovered in 1959
111 PTMab® citations

Acetylation (Ac)
Discovered in 1963
662 PTMab® citations

Ubiquitination (Ub)
Discovered in the 1980s
134 PTMab® citations

O-GlcNAcylation (O-GlcNAc)
Discovered in 1984

Nitration (nitro)
Discovered in the 1990s

SUMOylation (SUMO)
Discovered in the 1990s

Carboxyethylation (CEL)
Discovered in 1997

Propionylation (Pr)
Discovered in 2007

Butyrylation (Bu)
Discovered in 2007

Succinylation (Su)
Discovered in 2011
391 PTMab® citations

Malonylation (Mal) ★
Discovered in 2011
120 PTMab® citations

Crotonylation (Cr) ★
Discovered in 2011
265 PTMab® citations

Glutarylation (Glu) ★
Discovered in 2014

2-Hydryxoisobutyrylation (Hib) ★
Discovered in 2014

β-Hydroxybutyrylation (Bhb) ★
Discovered in 2016
100 PTMab® citations

Benzoylation (Bz) ★
Discovered in 2018

L-Lactylation (L-la) ★
Discovered in 2019
915 PTMab® citations

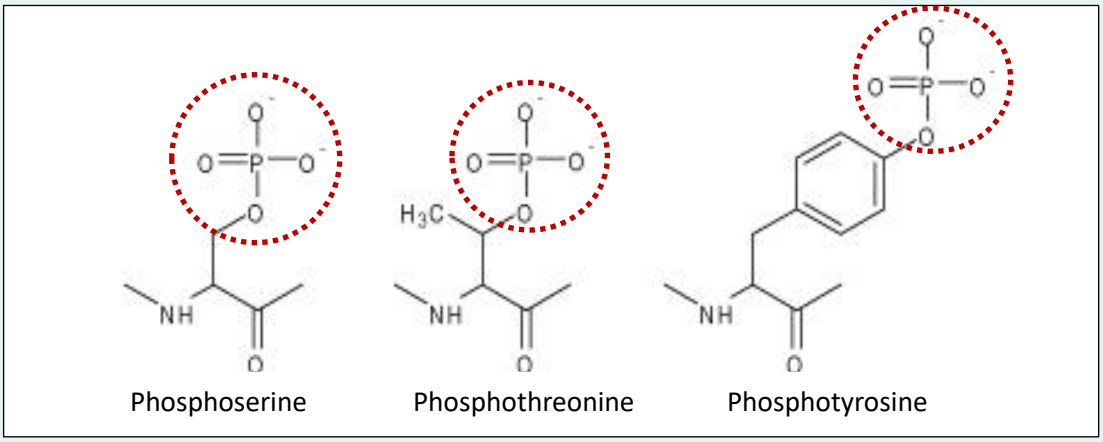
Methacrylation (Mea) ★
Discovered in 2021

D-Lactylation (D-la) ★
Discovered in 2024

★ **PTM Antibodies first developed by PTM BIO**

Phosphorylation

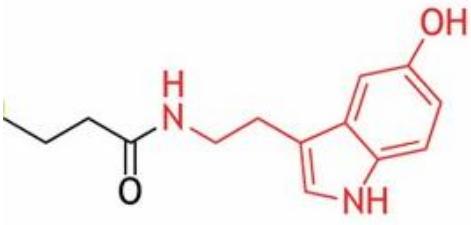
Protein phosphorylation was first recognized in the 1950s as a reversible regulatory process in which **phosphate** groups are covalently added to proteins. This modification typically occurs on **serine, threonine, or tyrosine** residues and is regulated by the opposing actions of protein kinases and phosphatases. As one of the most prevalent post-translational modifications (PTMs), phosphorylation is essential for regulating enzyme activity, receptor signaling, and numerous aspects of cellular communication, including signal transduction, development, differentiation, tumorigenesis, and stress responses. Dysregulation of phosphorylation is closely linked to diseases such as cancer, diabetes, and neurodegenerative disorders.



Cat #	Product Name	Applications	Citations
PTM-705RM	Anti-Phosphothreonine Rabbit mAb	WB, ICC/IF	6
PTM-730RM	Anti-Phosphothreonine Rabbit mAb (BSA and Azide Free)	WB	0
PTM-701	Anti-Phosphotyrosine Mouse mAb	WB	13
PTM-702RM	Anti-Phosphotyrosine Rabbit mAb	WB, IHC-P, IP	8
PTM-729RM	Anti-Phosphotyrosine Rabbit mAb (BSA and Azide Free)	WB	0
PTM-703	Anti-Phosphotyrosine Antibody Conjugated Agarose Beads	IAP	6
PTM-722	Anti-Phospho-Histone H1.4 (Thr146) Rabbit pAb	WB, IP	0
PTM-718	Anti-Phospho-Histone H2A (Thr120) Rabbit pAb	WB, IP	0
PTM-726	Anti-Phospho-Histone H2A (Ser129) Rabbit pAb	WB	0
PTM-753	Anti-Phospho-Histone H2A.X (Ser139) Mouse mAb	WB, IHC-P, ICC/IF	1
PTM-736	Anti-Phospho-Histone H2A.X (Ser139) Mouse mAb	WB, IP	0
PTM-727	Anti-Phospho-Histone H2A.X (Ser139) Rabbit mAb	WB, IHC-P	1
PTM-716	Anti-Phospho-Histone H2A.X (Ser139) Rabbit pAb	WB, IP	0
PTM-7224	Anti-Phospho-Histone H2B (Ser14) Rabbit mAb	WB	0
PTM-754	Anti-Phospho-Histone H2B (Tyr37) Mouse mAb	WB, IHC-P	0
PTM-710	Anti-Phospho-Histone H3 (Thr3) Rabbit pAb	WB, IP	0
PTM-757	Anti-Phospho-Histone H3 (Ser10) Mouse mAb	WB, IHC-P, ICC/IF	1
PTM-712RM	Anti-Phospho-Histone H3 (Ser10) Rabbit mAb	WB, IP, ChIP	0
PTM-712	Anti-Phospho-Histone H3 (Ser10) Rabbit pAb	WB, IHC-P, IP	0
PTM-743	Anti-Histone H3 (Trimethyl Lys9, Phospho Ser10) Mouse mAb	WB	0
PTM-723	Anti-Histone H3 (Trimethyl Lys9, Phospho Ser10) Rabbit pAb	WB, IHC-P	0
PTM-711	Anti-Phospho-Histone H3 (Thr11) Rabbit pAb	WB	0
PTM-756	Anti-Phospho-Histone H3 (Ser28) Mouse mAb	WB, ChIP	0
PTM-714	Anti-Phospho-Histone H3 (Ser28) Rabbit pAb	WB, IHC-P	0
PTM-720	Anti-Phospho-Histone H3 (Thr32) Rabbit pAb	WB	0
PTM-724RM	Anti-Phospho-Histone H3 (Thr45) Rabbit mAb	WB	0
PTM-724	Anti-Phospho-Histone H3 (Thr45) Rabbit pAb	WB	0

Serotonylation

Protein serotonylation, first described in the 1950s, is a post-translational modification in which **serotonin**, an excitatory neurotransmitter, is covalently attached to specific **glutamine** residues in target proteins by transglutaminases. It regulates key physiological processes, including platelet aggregation, insulin secretion, and pulmonary hypertension. In histone H3, serotonylation of Gln5 promotes the recruitment of TFIID, a multi-subunit complex that helps position RNA polymerase II at gene promoters to initiate transcription, acting in coordination with adjacent H3K4 trimethylation.

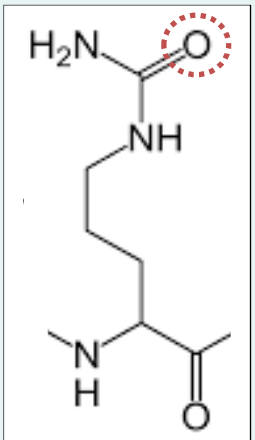


Glutamine Serotonylation

Cat #	Product Name	Applications	Citations
PTM-1420	Anti-Histone H3 (Trimethyl Lys4, Serotonyl Gln5) Rabbit pAb	WB, ChIP	1

Citrullination

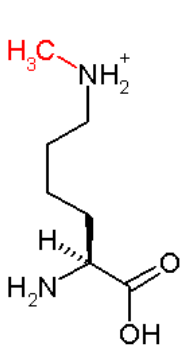
Protein citrullination, also known as deimination, is a post-translational modification in which the amino acid **arginine** is converted into **citrulline** by peptidylarginine deiminases (PADs). This reaction results in the loss of a positive charge and a slight increase in hydrophobicity, thereby altering protein structure, folding, and interactions. First described in the late 1950s, citrullination has since been recognized as a key regulator of physiological processes including epidermal differentiation, gene expression, and immune responses. Dysregulated citrullination is strongly implicated in disease, most notably in autoimmune disorders such as rheumatoid arthritis, where anti-citrullinated protein antibodies (ACPAs) serve as highly specific biomarkers, as well as in multiple sclerosis, cancer, and neurodegeneration

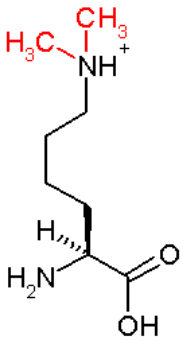


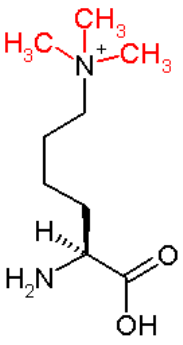
Citrullination

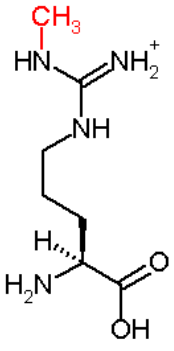
Cat #	Product Name	Applications	Citations
PTM-1306RM	Anti-Citrulline-Histone H3 (Arg2/8/17) Rabbit mAb	WB, IHC-P	0

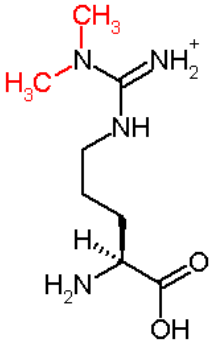
Methylation

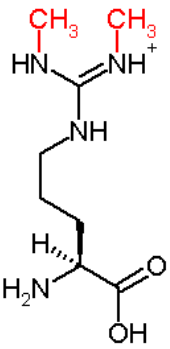

Monomethyllysine


Dimethyllysine


Trimethyllysine


Monomethylarginine


Asymmetric Dimethylarginine


Symmetric Dimethylarginine

Protein methylation, first reported in 1959, is a post-translational modification in which methyl groups are covalently added to **lysine** and **arginine** residues by methyltransferases. This modification can occur as **mono-, di-, or tri-methylation** on lysine and as **mono- or di-methylation** on arginine, altering protein structure, interactions, and function. Methylation plays critical roles in regulating gene expression, protein activity, RNA processing, and other epigenetic mechanisms, and has been implicated in cancer, aging, neurodegenerative diseases, and various other biological processes.

Lysine Methylation

Cat #	Product Name	Applications	Citations
PTM-602	Anti-Mono/Di-Methyl-Lysine Rabbit pAb	WB	13
PTM-606	Anti-Dimethyllysine Rabbit pAb	WB	7
PTM-606F	Anti-Dimethyllysine Rabbit mAb (BSA and Azide Free)	WB	0
PTM-601	Anti-Trimethyllysine Rabbit pAb	WB	12
PTM-698	Anti-Monomethyl-Histone H2B (Lys12) Rabbit pAb	WB, ChIP	0
PTM-661	Anti-Monomethyl-Histone H2B (Lys15) Mouse mAb	WB, IHC-P, IP, ChIP	0
PTM-5158	Anti-Monomethyl-Histone H3 (Lys4) Mouse mAb	WB, IHC-P, ChIP	1
PTM-611RM	Anti-Monomethyl-Histone H3 (Lys4) Rabbit mAb	WB, ICC/IF, IP	1
PTM-611	Anti-Monomethyl-Histone H3 (Lys4) Rabbit pAb	WB, IHC-P, ICC/IF	2
PTM-641	Anti-Dimethyl-Histone H3 (Lys4) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	2
PTM-6032	Anti-Dimethyl-Histone H3 (Lys4) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	1
PTM-5019	Anti-Trimethyl-Histone H3 (Lys4) Mouse mAb	WB, ChIP	4
PTM-613RM	Anti-Trimethyl-Histone H3 (Lys4) Rabbit mAb (ChIP Grade)	WB, FC, IP, ChIP	0
PTM-613	Anti-Trimethyl-Histone H3 (Lys4) Rabbit pAb	WB, IHC-P, ChIP	9
PTM-1420	Anti-Histone H3 (Trimethyl Lys4, Serotonyl Gln5) Rabbit pAb	WB, ChIP	1
PTM-7287	Anti-Monomethyl-Histone H3 (Lys9) Rabbit mAb	WB, ChIP	1
PTM-614	Anti-Monomethyl-Histone H3 (Lys9) Rabbit pAb	WB, ICC/IF, IP	2
PTM-5003	Anti-Dimethyl-Histone H3 (Lys9) Mouse mAb	WB, IHC-P, ICC/IF	0
PTM-644	Anti-Dimethyl-Histone H3 (Lys9) Mouse mAb	WB, IHC-P, ICC/IF, IP	2
PTM-615RM	Anti-Dimethyl-Histone H3 (Lys9) Rabbit mAb	WB, ICC/IF, IP, ChIP	1
PTM-616RM	Anti-Trimethyl-Histone H3 (Lys9) Rabbit mAb	WB, IHC-P, IP, ChIP	0
PTM-616	Anti-Trimethyl-Histone H3 (Lys9) Rabbit pAb	WB, IHC-P, ICC/IF	11
PTM-743	Anti-Histone H3 (Trimethyl Lys9, Phospho Ser10) Mouse mAb	WB	0
PTM-723	Anti-Histone H3 (Trimethyl Lys9, Phospho Ser10) Rabbit pAb	WB, IHC-P	0
PTM-640RM	Anti-Dimethyl-Histone H3 (Lys14) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	0
PTM-618RM	Anti-Monomethyl-Histone H3 (Lys18) Rabbit mAb	WB	0
PTM-632	Anti-Trimethyl-Histone H3 (Lys18) Rabbit pAb	WB, ChIP	0
PTM-619RM	Anti-Monomethyl-Histone H3 (Lys23) Rabbit mAb	WB, IHC-P, ICC/IF, IP, ChIP	0
PTM-619	Anti-Monomethyl-Histone H3 (Lys23) Rabbit pAb	WB, IP, ChIP	0
PTM-676	Anti-Dimethyl-Histone H3 (Lys23) Mouse mAb	WB, IHC-P, ICC/IF, IP, ChIP	0
PTM-648	Anti-Trimethyl-Histone H3 (Lys23) Mouse mAb	WB	0
PTM-5110	Anti-Monomethyl-Histone H3 (Lys27) Mouse mAb	WB, ChIP	1
PTM-620RM	Anti-Monomethyl-Histone H3 (Lys27) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	0
PTM-5312	Anti-Dimethyl-Histone H3 (Lys27) Mouse mAb	WB, IHC-P, ChIP	0
PTM-5010	Anti-Dimethyl-Histone H3 (Lys27) Rabbit mAb	WB, IHC-P, ICC/IF	2
PTM-651	Anti-Trimethyl-Histone H3 (Lys27) Mouse mAb	WB, IHC-P, ICC/IF	4
PTM-5002	Anti-Trimethyl-Histone H3 (Lys27) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	4
PTM-647RM	Anti-Trimethyl-Histone H3 (Lys27) Rabbit mAb	WB, IP, ChIP	1
PTM-623RM	Anti-Monomethyl-Histone H3 (Lys36) Rabbit mAb	WB, IHC-P, ICC/IF, IP	1

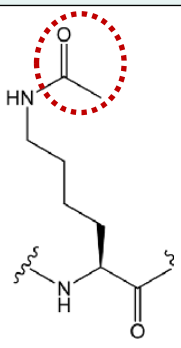
Cat #	Product Name	Applications	Citations
PTM-662	Anti-Dimethyl-Histone H3 (Lys36) Mouse mAb	WB, IHC-P, ChIP	0
PTM-5156	Anti-Dimethyl-Histone H3 (Lys36) Mouse mAb	WB, IHC-P	0
PTM-624	Anti-Dimethyl-Histone H3 (Lys36) Rabbit pAb	WB, IHC-P, ICC/IF	2
PTM-653RM	Anti-Trimethyl-Histone H3 (Lys36) Rabbit mAb	WB, IHC-P, IP, ChIP	0
PTM-625	Anti-Trimethyl-Histone H3 (Lys36) Rabbit pAb	WB, IHC-P, ICC/IF, IP	5
PTM-655	Anti-Monomethyl-Histone H3 (Lys56) Mouse mAb	WB, IP	0
PTM-669	Anti-Mono/Di-Methyl-Histone H3 (Lys56) Mouse mAb	WB	0
PTM-627RM	Anti-Dimethyl-Histone H3 (Lys56) Rabbit mAb	WB, IHC-P, ICC/IF, IP, ChIP	0
PTM-690	Anti-Trimethyl-Histone H3 (Lys64) Rabbit pAb	WB, ChIP	0
PTM-5112	Anti-Monomethyl-Histone H3 (Lys79) Rabbit mAb	WB, IHC-P	1
PTM-628RM	Anti-Monomethyl-Histone H3 (Lys79) Rabbit mAb	WB, IP, ChIP	0
PTM-628	Anti-Monomethyl-Histone H3 (Lys79) Rabbit pAb	WB, IHC-P, ICC/IF, IP	2
PTM-657RM	Anti-Mono/Di-Methyl-Histone H3 (Lys79) Rabbit mAb	WB, IHC-P, ICC/IF, FC, IP, ChIP	0
PTM-7435	Anti-Mono/Di/Tri-Methyl-Histone H3 (Lys79) Rabbit mAb	WB, IHC-P	0
PTM-5159	Anti-Dimethyl-Histone H3 (Lys79) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	2
PTM-658	Anti-Dimethyl-Histone H3 (Lys79) Mouse mAb	WB, IHC-P, ICC/IF, IP	0
PTM-629	Anti-Dimethyl-Histone H3 (Lys79) Rabbit pAb	WB, IHC-P, IP	2
PTM-5157	Anti-Trimethyl-Histone H3 (Lys79) Mouse mAb	WB, IHC-P	0
PTM-689	Anti-Trimethyl-Histone H3 (Lys79) Mouse mAb	WB, ChIP	0
PTM-631RM	Anti-Monomethyl-Histone H3 (Lys122) Rabbit mAb	WB	0
PTM-645RM	Anti-Dimethyl-Histone H3 (Lys122) Rabbit mAb	WB, ICC/IF, IP, ChIP	0
PTM-685RM	Anti-Monomethyl-Histone H4 (Lys12) Rabbit mAb	WB, IHC-P, ICC/IF, FC, IP, ChIP	2
PTM-5111	Anti-Monomethyl-Histone H4 (Lys20) Mouse mAb	WB, ICC/IF, ChIP	0
PTM-634	Anti-Monomethyl-Histone H4 (Lys20) Rabbit pAb	WB, ChIP	0
PTM-5005	Anti-Trimethyl-Histone H4 (Lys20) Mouse mAb	WB	0
PTM-636	Anti-Trimethyl-Histone H4 (Lys20) Rabbit pAb	WB, IHC-P	0
PTM-637	Anti-Monomethyl-Histone H4 (Lys31) Rabbit pAb	WB	0

Arginine Methylation

Cat #	Product Name	Applications	Citations
PTM-605RM	Anti-Asymmetric Dimethylarginine Rabbit mAb	WB	3
PTM-617RM	Anti-Symmetric Dimethylarginine Rabbit mAb	WB, IHC-P, ICC/IF	2
PTM-688	Anti-Monomethyl-Histone H2A/H4 (Arg3) Rabbit pAb	WB	0
PTM-660	Anti-Asymmetric Dimethyl-Histone H2A/H4 (Arg3) Mouse mAb	WB	0
PTM-673	Anti-Symmetric Dimethyl-Histone H2A (Arg3) Rabbit pAb	WB	0
PTM-700	Anti-Monomethyl-Histone H3 (Arg2) Rabbit mAb	WB, ICC/IF	0
PTM-633	Anti-Monomethyl-Histone H3 (Arg2) Rabbit pAb	WB, IP	0
PTM-668	Anti-Asymmetric Dimethyl-Histone H3 (Arg2) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	2
PTM-671	Anti-Asymmetric Dimethyl-Histone H3 (Arg2) Rabbit pAb	WB, ChIP	0
PTM-652RM	Anti-Symmetric Dimethyl-Histone H3 (Arg2) Rabbit mAb	WB, ChIP	0
PTM-695	Anti-Monomethyl-Histone H3 (Arg8) Mouse mAb	WB, IHC-P, ICC/IF, IP	0
PTM-650RM	Anti-Asymmetric Dimethyl-Histone H3 (Arg17) Rabbit mAb	WB, ChIP	1
PTM-667	Anti-Asymmetric Dimethyl-Histone H4 (Arg3) Mouse mAb	WB, IHC-P, ChIP	1
PTM-699	Anti-Monomethyl-Histone H4 (Arg19) Rabbit pAb	WB	0
PTM-694	Anti-Monomethyl-Histone H4 (Arg23) Rabbit pAb	WB	0

Acetylation

Originally discovered in 1963 as a unique modification on histone proteins, acetylation marks are now recognized as a widespread and critical post-translational modification. It involves the covalent addition of an **acetyl** group to **lysine** residues or to the protein **N-terminus**, catalyzed by acetyltransferases and removed by deacetylases, thereby dynamically regulating protein charge, stability, localization, and interactions. Histone acetylation, tightly controlled by the opposing action of histone acetyltransferases (HATs) and histone deacetylases (HDACs), occurs primarily at lysine residues on the N-terminal tails of histones H2A (Lys5, 9, and 15), H2B (Lys5, 12, 15, 16, and 20), H3 (Lys4, 9, 14, 18, 23, 27, and 36), and H4 (Lys5, 8, 12, 16, and 20). This modification plays vital roles in the regulation of gene expression, DNA damage repair, and chromatin dynamics. Dysregulation of acetylation is implicated in cancer, neurodegenerative diseases, cardiovascular disorders, and aging, making it a central focus in epigenetics and therapeutic research.



Lysine Acetylation

Cat #	Product Name	Applications	Citations
PTM-101	Anti-Acetyllysine Mouse mAb	WB, IHC-P	127
PTM-102	Anti-Acetyllysine Mouse mAb	WB, ICC/IF	23
PTM-105RM	Anti-Acetyllysine Rabbit mAb	WB, IHC-P	54
PTM-104	Anti-Acetyllysine Antibody Conjugated Agarose Beads	IAP	205
PTM-177	Anti-Acetyl-Histone H1.4 (Lys25) Rabbit pAb	WB	0
PTM-106RM	Anti-Acetyl-Histone H2A (Lys5) Rabbit mAb	WB, ChIP	0
PTM-106	Anti-Acetyl-Histone H2A (Lys5) Rabbit pAb	WB	3
PTM-193	Anti-Acetyl-Histone H2A (Lys9) Mouse mAb	WB	1
PTM-194	Anti-Acetyl-Histone H2A (Lys9) Rabbit mAb	WB, IP, ChIP	0
PTM-175	Anti-Acetyl-Histone H2A (Lys15) Mouse mAb	WB, IHC-P	0
PTM-172	Anti-Acetyl-Histone H2A (Lys15) Rabbit pAb	WB	0
PTM-173	Anti-Acetyl-Histone H2A (Lys118) Rabbit pAb	WB	1
PTM-152	Anti-Acetyl-Histone H2B (Lys5) Mouse mAb	WB, IHC-P, IP, ChIP	0
PTM-107	Anti-Acetyl-Histone H2B (Lys5) Rabbit pAb	WB, IHC-P	0
PTM-176	Anti-Acetyl-Histone H2B (Lys11) Mouse mAb	WB, IHC-P, ICC/IF, IP, ChIP	0
PTM-130	Anti-Acetyl-Histone H2B (Lys11) Rabbit pAb	WB	1
PTM-153	Anti-Acetyl-Histone H2B (Lys12) Mouse mAb	WB, IHC-P, ChIP	1
PTM-108	Anti-Acetyl-Histone H2B (Lys12) Rabbit pAb	WB, ChIP	1
PTM-181	Anti-Acetyl-Histone H2B (Lys15) Mouse mAb	WB, IHC-P	0
PTM-109	Anti-Acetyl-Histone H2B (Lys15) Rabbit pAb	WB, IHC-P	0
PTM-167	Anti-Acetyl-Histone H2B (Lys16) Mouse mAb	WB, IHC-P, ICC/IF	0
PTM-123	Anti-Acetyl-Histone H2B (Lys16) Rabbit pAb	WB, IHC-P, ChIP	0
PTM-155	Anti-Acetyl-Histone H2B (Lys20) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	1
PTM-110	Anti-Acetyl-Histone H2B (Lys20) Rabbit pAb	WB, IHC-P, ChIP	0
PTM-174	Anti-Acetyl-Histone H2B (Lys23) Mouse mAb	WB	2
PTM-171	Anti-Acetyl-Histone H2B (Lys23) Rabbit pAb	WB, ChIP	1
PTM-169	Anti-Acetyl-Histone H2B (Lys24) Mouse mAb	WB, ICC/IF, ChIP	0
PTM-126	Anti-Acetyl-Histone H2B (Lys24) Rabbit pAb	WB	1
PTM-182RM	Anti-Acetyl-Histone H2B (Lys46) Rabbit mAb	WB, ICC/IF, ChIP	0
PTM-182	Anti-Acetyl-Histone H2B (Lys46) Rabbit pAb	WB	0
PTM-111	Anti-Acetyl-Histone H2B (Lys120) Rabbit pAb	WB, ChIP	1
PTM-168	Anti-Acetyl-Histone H3 (Lys4) Mouse mAb	WB, ICC/IF	2

Cat #	Product Name	Applications	Citations
PTM-188	Anti-Acetyl-Histone H3 (Lys4) Rabbit mAb	WB, IHC-P, ICC/IF	0
PTM-124	Anti-Acetyl-Histone H3 (Lys4) Rabbit pAb	WB, ChIP	0
PTM-156	Anti-Acetyl-Histone H3 (Lys9) Mouse mAb	WB, IP, ChIP	11
PTM-112RM	Anti-Acetyl-Histone H3 (Lys9) Rabbit mAb	WB, IHC-P, ChIP, CUT&Tag	6
PTM-112	Anti-Acetyl-Histone H3 (Lys9) Rabbit pAb	WB, IHC-P, IP	12
PTM-157	Anti-Acetyl-Histone H3 (Lys14) Mouse mAb	WB, IHC-P, ICC/IF, IP, ChIP	9
PTM-113RM	Anti-Acetyl-Histone H3 (Lys14) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	5
PTM-158	Anti-Acetyl-Histone H3 (Lys18) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	13
PTM-114RM	Anti-Acetyl-Histone H3 (Lys18) Rabbit mAb	WB, IHC-P, ChIP	8
PTM-115RM	Anti-Acetyl-Histone H3 (Lys23) Rabbit mAb	WB, IHC-P, IP, ChIP	2
PTM-115	Anti-Acetyl-Histone H3 (Lys23) Rabbit pAb	WB	3
PTM-160	Anti-Acetyl-Histone H3 (Lys27) Mouse mAb	WB, IHC-P, ICC/IF	11
PTM-116RM	Anti-Acetyl-Histone H3 (Lys27) Rabbit mAb	WB, IHC-P, ICC/IF, IP, ChIP	6
PTM-116	Anti-Acetyl-Histone H3 (Lys27) Rabbit pAb	WB, IHC-P, ChIP	6
PTM-117RM	Anti-Acetyl-Histone H3 (Lys36) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	1
PTM-117	Anti-Acetyl-Histone H3 (Lys36) Rabbit pAb	WB, IHC-P, ICC/IF, ChIP	3
PTM-128	Anti-Acetyl-Histone H3 (Lys37) Rabbit pAb	WB	0
PTM-162	Anti-Acetyl-Histone H3 (Lys56) Mouse mAb	WB, ICC/IF, ChIP	5
PTM-118	Anti-Acetyl-Histone H3 (Lys56) Rabbit pAb	WB, IP, ChIP	4
PTM-129RM	Anti-Acetyl-Histone H3 (Lys64) Rabbit mAb	WB, IHC-P, ChIP	0
PTM-129	Anti-Acetyl-Histone H3 (Lys64) Rabbit pAb	WB	0
PTM-170	Anti-Acetyl-Histone H3 (Lys115) Rabbit pAb	WB	2
PTM-184RM	Anti-Acetyl-Histone H3 (Lys122) Rabbit mAb	WB, IHC-P	1
PTM-184	Anti-Acetyl-Histone H3 (Lys122) Rabbit pAb	WB	1
PTM-163	Anti-Acetyl-Histone H4 (Lys5) Mouse mAb	WB, IHC-P, ICC/IF	11
PTM-119	Anti-Acetyl-Histone H4 (Lys5) Rabbit pAb	WB, IHC-P, ICC/IF, ChIP	7
PTM-189	Anti-Acetyl-Histone H4 (Lys5/8/12/16) Mouse mAb	WB, IHC-P, ICC/IF	1
PTM-164	Anti-Acetyl-Histone H4 (Lys8) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	10
PTM-120	Anti-Acetyl-Histone H4 (Lys8) Rabbit pAb	WB, IHC-P, ICC/IF	9
PTM-165	Anti-Acetyl-Histone H4 (Lys12) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	9
PTM-192	Anti-Acetyl-Histone H4 (Lys12) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	2
PTM-121RM	Anti-Acetyl-Histone H4 (Lys12) Rabbit mAb	WB, IHC-P	1
PTM-121	Anti-Acetyl-Histone H4 (Lys12) Rabbit pAb	WB, IHC-P, ICC/IF	2
PTM-187	Anti-Acetyl-Histone H4 (Lys16) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	2
PTM-122	Anti-Acetyl-Histone H4 (Lys16) Rabbit pAb	WB, IHC-P, ICC/IF, ChIP	14
PTM-127RM	Anti-Acetyl-Histone H4 (Lys77) Rabbit mAb	WB, IHC-P, IP, ChIP	0

Ubiquitination

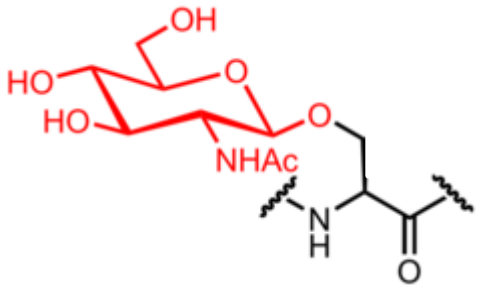
Ubiquitination, discovered in the 1980s, is a highly conserved post-translational modification in which the 76-amino acid protein **ubiquitin** is covalently attached to **lysine** residues on substrate proteins. This process is mediated by a three-step enzymatic cascade involving ubiquitin-acting enzymes (E1), ubiquitin-conjugating enzymes (E2), and ubiquitin ligases (E3), which together confer specificity and regulatory control. Ubiquitination proceeds through the formation of an isopeptide bond between its C-terminal Gly76 of ubiquitin and the ε-amino group of a target protein lysine residue.

Ubiquitination can occur as monoubiquitin, in which a single ubiquitin is attached, or as polyubiquitin chains, where additional ubiquitin molecules are linked to one another through either the N-terminus (M1) or one of seven Lys residues (Lys6, Lys11, Lys27, Lys29, Lys33, Lys48, and Lys63). These linkages encode distinct cellular signals. Lys6-linked chains have been implicated in DNA repair, Lys11-linked chains in endoplasmic reticulum-associated degradation (ERAD) and cell-cycle regulation, Lys29-linked chains in lysosomal degradation, Lys33-linked chains in kinase modification, Lys48-linked chains in proteasomal degradation, and Lys63-linked chains in endocytosis and DNA damage responses.

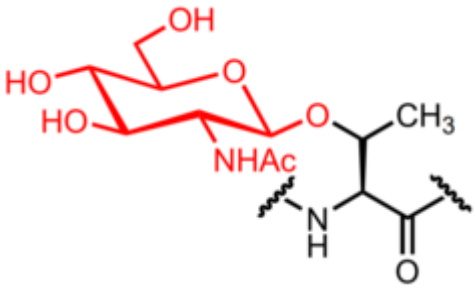
Cat #	Product Name	Applications	Citations
PTM-5798	Anti-Ubiquitin Mouse mAb (N-terminal)	WB, ICC/IF	6
PTM-1107	Anti-Ubiquitin Mouse mAb (N-terminal)	WB, IHC-P	21
PTM-1106RM	Anti-Ubiquitin Rabbit mAb (N-terminal)	WB, ICC/IF	22
PTM-1124RM	Anti-Ubiquitin Rabbit mAb (N-terminal, BSA and Azide Free)	WB	1
PTM-1104	Anti-Diglycyllysine Antibody Conjugated Agarose Beads	IAP	60
PTM-1121	Anti-Ubiquityl-Histone H2A (Lys119) Rabbit mAb	WB, IHC-P, ICC/IF, IP, ChIP	4
PTM-1122	Anti-Ubiquityl-Histone H2B (Lys120) Mouse mAb	WB, IHC-P	0
PTM-1123RM	Anti-Ubiquityl-Histone H2B (Lys120) Rabbit mAb	WB, IHC-P	1

O-GlcNAcylation

O-linked β-N-acetylglucosamine modification (O-GlcNAcylation), first reported in 1984, is a unique form of glycosylation in which the monosaccharide **β-N-acetylglucosamine (GlcNAc)** is covalently attached to **serine** or **threonine** residues via an O-linked glycosidic bond. Unlike classical glycosylation, O-GlcNAcylation occurs primarily in the cytoplasm and nucleoplasm. This dynamic modification is regulated by two enzymes: O-GlcNAc transferase (OGT), which catalyzes the attachment of GlcNAc, and O-GlcNAcase (OGA, β-N-acetylglucosaminidase), which removes it. O-GlcNAcylation has been identified on histones—including H2A (T101), H2B (S36, S112), H3 (S10, T32), and H4 (S47)—highlighting its role in chromatin regulation. Because its donor substrate, UDP-GlcNAc, is a metabolic product of the hexosamine biosynthetic pathway, O-GlcNAcylation functions as a nutrient sensor that couples cellular metabolism to signaling and gene expression. Dysregulation of this modification has been implicated in cancer, diabetes, cardiovascular disease, and neurodegenerative disorders.



Serine O-GlcNAcylation



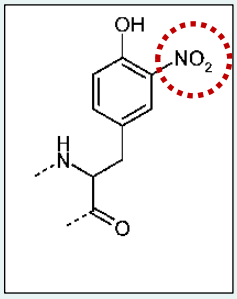
Threonine O-GlcNAcylation

3-Nitrotyrosine

Cat #	Product Name	Applications	Citations
PTM-951RM	Anti-O-Linked N-Acetylglucosamine Rabbit mAb	WB, IHC-P, ICC/IF	6
PTM-955RM	Anti-O-Linked N-Acetylglucosamine Rabbit mAb (BSA and Azide Free)	WB	0

Nitration

Protein tyrosine nitration, biologically recognized in the early 1990s, involves the covalent addition of a **nitro group** (-NO_2) to **tyrosine**, forming 3-nitrotyrosine. This modification is mediated by reactive nitrogen species (RNS) such as peroxynitrite anion ($ONOO^-$) and nitrogen dioxide ($\bullet NO_2$), which arise under oxidative and nitrosative stress. Aberrant protein nitration has been implicated in cardiovascular disease, cancer, diabetes, and neurodegenerative disorders such as Alzheimer's and Parkinson's disease.



Cat #	Product Name	Applications	Citations
PTM-751	Anti-3-Nitrotyrosine Rabbit pAb	WB	0

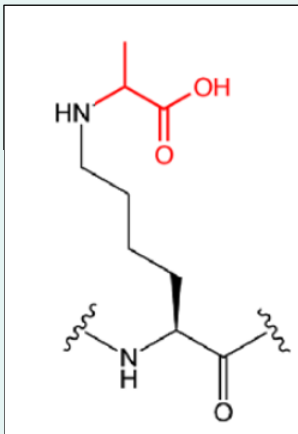
SUMOylation

First described in the 1990s, SUMOylation is a reversible post-translational modification in which **Small Ubiquitin-like Modifier** (SUMO) proteins are covalently attached to target proteins. SUMO belongs to the ubiquitin-like protein family, with three primary human isoforms (SUMO-1, SUMO-2, and SUMO-3). SUMO attaches via an isopeptide bond to **lysine** residues of substrates, functioning as either a monomer or in lysine-linked polymeric chains. SUMO-1 modification regulates key proteins such as RanGAP1, PML, p53, and IκB-α, impacting nuclear trafficking, transcriptional activity, and protein turnover. SUMO-2/3, which readily form poly-SUMO chains, modify proteins such as topoisomerase II and APP, playing roles in genome stability and cellular responses to environmental stress. Dysregulation of this modification has been linked to cardiovascular diseases, cancer, neurodegenerative disorders, and immune-related pathologies.

Cat #	Product Name	Applications	Citations
PTM-1109	Anti-SUMO-1/2/3 Mouse mAb (N-terminal)	WB, IHC-P	4
PTM-1104	Anti-Diglycyllysine Antibody Conjugated Agarose Beads	IAP	60

Carboxyethylation

Lysine carboxyethylation, most commonly represented by N^{ε}-(carboxyethyl)lysine (CEL), is a stable, nonenzymatic protein modification. First described in 1997, CEL forms through the reaction of **lysine** residues with **methylglyoxal**, a byproduct of glucose and lipid metabolism. This modification can alter the structure and function of proteins, affecting their activity, stability, and interactions. Accumulation of CEL is closely associated with metabolic stress and disease conditions. Elevated CEL levels have been reported in diabetic complications, atherosclerosis, disorders, and neurodegenerative diseases.

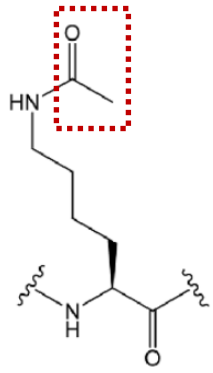


N^{ε}-(carboxyethyl)lysine

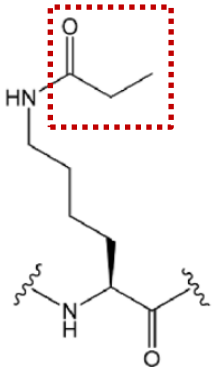
Cat #	Product Name	Applications	Citations
PTM-1701RM	Anti-Carboxyethyllysine Rabbit mAb	WB	4

Propionylation

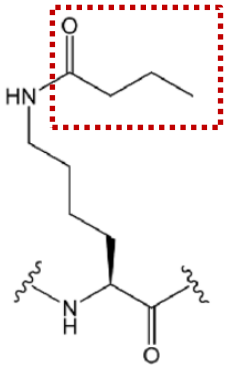
First reported in 2007, lysine propionylation is a reversible post-translational modification in which a **propionyl** group (–CO–CH_2–CH_3) is covalently attached to the ε-amino group of **lysine** residues. This modification has been well documented in both prokaryotes and eukaryotes, affecting a wide range of proteins, including histones and non-histone substrates such as p53. It is speculated that lysine propionylation plays a vital role in the regulation of multiple cellular processes including chromatin dynamics, plasticity, and DNA transcriptional regulation by sharing same regulative enzymes with lysine acetylation or with its unique regulative enzymes.



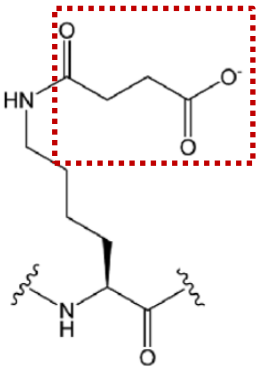
Acetyllysine



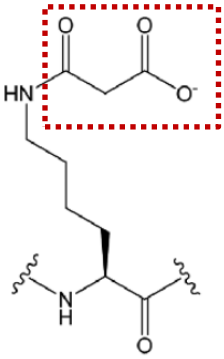
Propionyllysine



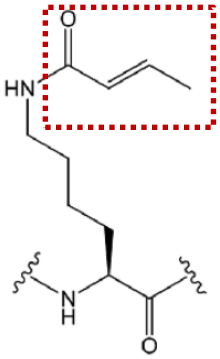
Butyryllysine



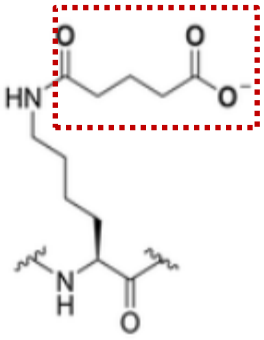
Succinyllysine



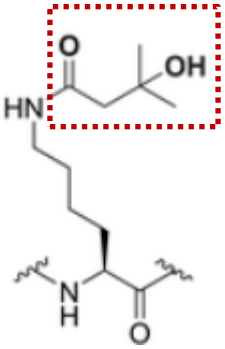
Malonyllysine



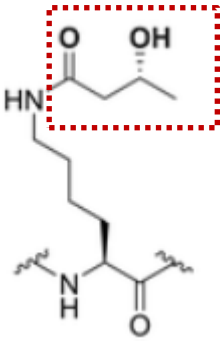
Crotonyllysine



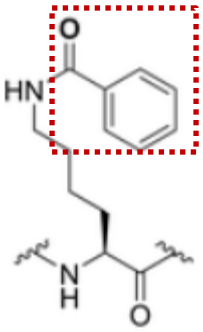
Glutaryllysine



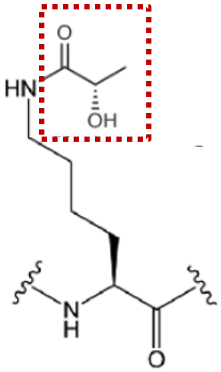
2-Hydroxyisobutyryllysine



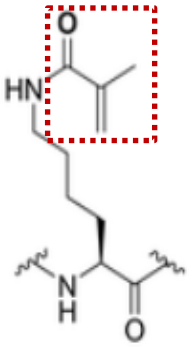
β-Hydroxybutyryllysine



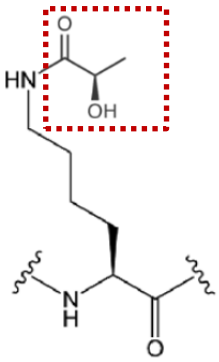
Benzoyllysine



L-lactyllysine



Methacryllysine



D-lactyllysine

Cat #	Product Name	Applications	Citations
PTM-203	Anti-Propionyllysine Mouse mAb	WB, IHC-P	5
PTM-201	Anti-Propionyllysine Rabbit pAb	WB	25
PTM-202	Anti-Propionyllysine Antibody Conjugated Agarose Beads	IAP	14
PTM-217RM	Anti-Propionyl-Histone H2A (Lys125) Rabbit mAb	WB	0
PTM-217	Anti-Propionyl-Histone H2A (Lys125) Rabbit pAb	WB	0
PTM-215	Anti-Propionyl-Histone H3 (Lys14) Mouse mAb	WB	2
PTM-211	Anti-Propionyl-Histone H3 (Lys14) Rabbit pAb	WB	1
PTM-214	Anti-Propionyl-Histone H3 (Lys18) Mouse mAb	WB	0
PTM-213	Anti-Propionyl-Histone H3 (Lys18) Rabbit pAb	WB	5
PTM-205	Anti-Propionyl-Histone H3 (Lys23) Rabbit pAb	WB, IHC-P	4
PTM-220RM	Anti-Propionyl-Histone H3 (Lys56) Rabbit mAb	WB, IHC-P	0
PTM-216	Anti-Propionyl-Histone H4 (Lys5) Mouse mAb	WB, IHC-P	0
PTM-219RM	Anti-Propionyl-Histone H4 (Lys5) Rabbit mAb	WB, IHC-P, ICC/IF, IP	0
PTM-218	Anti-Propionyl-Histone H4 (Lys8) Mouse mAb	WB, IHC-P, ChIP	0
PTM-209	Anti-Propionyl-Histone H4 (Lys12) Mouse mAb	WB, IHC-P	2
PTM-206	Anti-Propionyl-Histone H4 (Lys12) Rabbit pAb	WB, ICC/IF, ChIP	0
PTM-210	Anti-Propionyl-Histone H4 (Lys16) Mouse mAb	WB, IHC-P, ChIP	5

Butyrylation

First reported in 2007, lysine butyrylation is a reversible post-translational modification in which a **butyryl** group (–CO–CH_2–CH_2–CH_3) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). Structurally similar to lysine acetylation and lysine propionylation, this modification has emerged as an important regulator of protein function. Many lysine residues in both histones and non-histone substrates, such as p53 and p300/CBP, have been identified as butyrylation sites, suggesting that this modification plays a critical role in epigenetic regulation. In particular, lysine butyrylation influences chromatin dynamics and plasticity, DNA transcriptional regulation, and tumorigenesis.

Cat #	Product Name	Applications	Citations
PTM-329	Anti-Butyryllysine Mouse mAb	WB, ICC/IF	3
PTM-301RM	Anti-Butyryllysine Rabbit mAb	WB, IHC-P, ICC/IF	1
PTM-337RM	Anti-Butyryllysine Rabbit mAb (BSA and Azide Free)	WB	0
PTM-302	Anti-Butyryllysine Antibody Conjugated Agarose Beads	IAP	4
PTM-327	Anti-Butyryl-Histone H2A (Lys5) Mouse mAb	WB, ChIP	0
PTM-317	Anti-Butyryl-Histone H2A (Lys5) Rabbit pAb	WB, IHC-P, ICC/IF	0
PTM-320	Anti-Butyryl-Histone H2B (Lys5) Mouse mAb	WB, IHC-P, ChIP	0
PTM-303	Anti-Butyryl-Histone H2B (Lys5) Rabbit pAb	WB, IHC-P, ICC/IF	0
PTM-323	Anti-Butyryl-Histone H2B (Lys12) Mouse mAb	WB	0
PTM-318	Anti-Butyryl-Histone H2B (Lys12) Rabbit pAb	WB	0
PTM-324	Anti-Butyryl-Histone H2B (Lys15) Mouse mAb	WB, ICC/IF	0
PTM-333	Anti-Butyryl-Histone H2B (Lys15) Rabbit pAb	WB, ICC/IF, ChIP	0
PTM-322	Anti-Butyryl-Histone H2B (Lys16) Mouse mAb	WB, ICC/IF, ChIP	0
PTM-319	Anti-Butyryl-Histone H2B (Lys16) Rabbit pAb	WB, IHC-P, ICC/IF	0
PTM-316	Anti-Butyryl-Histone H2B (Lys20) Mouse mAb	WB, IHC-P	0
PTM-304	Anti-Butyryl-Histone H2B (Lys20) Rabbit pAb	WB, IHC-P, ICC/IF	0
PTM-335RM	Anti-Butyryl-Histone H2B (Lys23) Rabbit mAb	WB	0
PTM-312	Anti-Butyryl-Histone H3 (Lys9) Mouse mAb	WB, ICC/IF, ChIP	0

Cat #	Product Name	Applications	Citations
PTM-334RM	Anti-Butyryl-Histone H3 (Lys14) Rabbit mAb	WB	0
PTM-331	Anti-Butyryl-Histone H3 (Lys18) Mouse mAb	WB, ChIP	1
PTM-338RM	Anti-Butyryl-Histone H3 (Lys18) Rabbit mAb	WB, IP, ChIP	0
PTM-307	Anti-Butyryl-Histone H3 (Lys23) Mouse mAb	WB, IHC-P	3
PTM-326	Anti-Butyryl-Histone H3 (Lys27) Mouse mAb	WB, ICC/IF	0
PTM-315	Anti-Butyryl-Histone H3 (Lys27) Rabbit pAb	WB, ChIP	1
PTM-336	Anti-Butyryl-Histone H3 (Lys115) Rabbit pAb	WB	0
PTM-310	Anti-Butyryl-Histone H4 (Lys5) Mouse mAb	WB, IHC-P, ChIP	2
PTM-313	Anti-Butyryl-Histone H4 (Lys5) Rabbit pAb	WB	3
PTM-311	Anti-Butyryl-Histone H4 (Lys8) Rabbit pAb	WB, ICC/IF, ChIP	3
PTM-314	Anti-Butyryl-Histone H4 (Lys12) Mouse mAb	WB	1

Succinylation

Lysine succinylation is a reversible post-translational modification in which a **succinyl** group (–CO–CH_2–CH_2–CO_2H) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). First described in 2011, this modification has been identified in a wide range of proteins, including histones and key metabolic enzymes. A defining feature of succinylation is that it alters lysine's charge from +1 to –1 at physiological pH and introduces a relatively bulky moiety of ~100 Da. In comparison, acetylation adds only 42 Da and methylation 14 Da. Because of this significant size and charge shift, succinylation is expected to induce more substantial changes in protein structure, stability, and function than many other lysine modifications.

The regulatory machinery of succinylation includes "writers" such as carnitine palmitoyltransferase 1A (CPT1A) and lysine acetyltransferase 2A (KAT2A/GCN5); "erasers" like sirtuin 5 (SIRT5) and sirtuin 7 (SIRT7); and a pH-dependent "reader," the YEATS domain of glioma-amplified sequence 41 (GAS41). Together, these proteins control the dynamic installation, removal, and recognition of succinyl groups. Although the precise biological consequences of succinylation remain under investigation, current evidence links this modification to chromatin regulation, cellular metabolism, and stress responses.

Cat #	Product Name	Applications	Citations
PTM-419	Anti-Succinyllysine Mouse mAb	WB	95
PTM-401	Anti-Succinyllysine Rabbit pAb	WB	186
PTM-402	Anti-Succinyllysine Antibody Conjugated Agarose Beads	IAP	91
PTM-409	Anti-Succinyl-Histone H2B (Lys120) Rabbit pAb	WB	2
PTM-421	Anti-Succinyl-Histone H3 (Lys14) Mouse mAb	WB, IHC-P	1
PTM-422	Anti-Succinyl-Histone H3 (Lys23) Mouse mAb	WB, IHC-P	1
PTM-412	Anti-Succinyl-Histone H3 (Lys79) Rabbit pAb	WB	7
PTM-413	Anti-Succinyl-Histone H3 (Lys122) Rabbit pAb	WB	8

Malonylation

Lysine malonylation is a reversible post-translational modification (PTM) first reported in 2011 that regulates protein activity and function. This modification involves the covalent attachment of a **malonyl** group (–CO–CH_2–COOH) to the ε-amino group of **lysine** residues (see structure on page 10). By shifting lysine's charge from +1 to –1 at physiological pH and adding a bulky moiety of ~86 Da, malonylation induces more pronounced structural and electrostatic changes than smaller modifications such as acetylation (42 Da) or methylation (14 Da).

Malonylation is dynamically regulated by enzymes, with sirtuin 5 (SIRT5) identified as a key demalonylase capable of catalyzing lysine demalonylation both in vivo and in vitro. Importantly, lysine malonylation has been closely linked to metabolic control, particularly through its dependence on malonyl-CoA, a central metabolite in fatty acid biosynthesis.

Cat #	Product Name	Applications	Citations
PTM-902	Anti-Malonyllysine Mouse mAb	WB	17
PTM-901	Anti-Malonyllysine Rabbit pAb	WB	76
PTM-904	Anti-Malonyllysine Antibody Conjugated Agarose Beads	IAP	27
PTM-911RM	Anti-Malonyl-Histone H3 (Lys23) Rabbit mAb	WB, ICC/IF, ChIP	0
PTM-910RM	Anti-Malonyl-Histone H3 (Lys122) Rabbit mAb	WB, IHC-P	0
PTM-910	Anti-Malonyl-Histone H3 (Lys122) Rabbit pAb	WB	0

Crotonylation

Protein crotonylation is a reversible post-translational modification first identified in 2011. It involves the covalent attachment of a **crotonyl** group (–CO–CH=CH–CH_3) to **lysine** (see structure on page 10) and, less commonly, **serine** residues. The crotonyl group introduces a planar, unsaturated four-carbon moiety that alters the local charge distribution and hydrophobicity of proteins, potentially affecting their confirmation, interactions, and functions.
Crotonylation is dynamically regulated by specific “writers,” “erasers,” and “readers.” Histone acetyltransferases such as p300/CBP have been shown to function as crotonyltransferases, while histone deacetylases (HDACs) and sirtuins act as decrotonylases. YEATS domain–containing proteins serve as specialized “readers” that recognize crotonyl-lysine marks with high affinity, linking this modification to downstream regulatory outcomes. Crotonylation has been linked to transcriptional activation in specific contexts, including spermatogenesis, early embryonic development, and cellular stress responses.

Cat #	Product Name	Applications	Citations
PTM-502	Anti-Crotonyllysine Mouse mAb	WB	74
PTM-501	Anti-Crotonyllysine Rabbit pAb	WB	69
PTM-503	Anti-Crotonyllysine Antibody Conjugated Agarose Beads	IAP	45
PTM-543	Anti-Crotonyl-Histone H2A (Lys119) Rabbit mAb	WB, ICC/IF	1
PTM-505	Anti-Crotonyl-Histone H2A (Lys119) Rabbit pAb	WB, ChIP	0
PTM-508RM	Anti-Crotonyl-Histone H2B (Lys11) Rabbit mAb	WB, IHC-P	0
PTM-508	Anti-Crotonyl-Histone H2B (Lys11) Rabbit pAb	WB	1
PTM-528	Anti-Crotonyl-Histone H2B (Lys12) Mouse mAb	WB, IHC-P, ChIP	0
PTM-509	Anti-Crotonyl-Histone H2B (Lys12) Rabbit pAb	WB, ChIP	4
PTM-546RM	Anti-Crotonyl-Histone H2B (Lys15/16/20) Rabbit mAb	WB, ChIP	0
PTM-533	Anti-Crotonyl-Histone H2B (Lys16) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	0
PTM-534	Anti-Crotonyl-Histone H2B (Lys20) Mouse mAb	WB, ChIP	0
PTM-512	Anti-Crotonyl-Histone H2B (Lys20) Rabbit pAb	WB, IP	2
PTM-514	Anti-Crotonyl-Histone H2B (Lys34) Rabbit pAb	WB, ChIP	3
PTM-527	Anti-Crotonyl-Histone H3 (Lys4) Mouse mAb	WB, ChIP	5
PTM-515RM	Anti-Crotonyl-Histone H3 (Lys4) Rabbit mAb	WB, IHC-P, ICC/IF	2
PTM-539	Anti-Crotonyl-Histone H3 (Lys9) Mouse mAb	WB, ChIP	1
PTM-516RM	Anti-Crotonyl-Histone H3 (Lys9) Rabbit mAb	WB, IHC-P, ICC/IF, IP	3
PTM-537	Anti-Crotonyl-Histone H3 (Lys14) Mouse mAb	WB, ICC/IF, IP, ChIP	0
PTM-535RM	Anti-Crotonyl-Histone H3 (Lys14) Rabbit mAb	WB, IHC-P, ChIP	0
PTM-535	Anti-Crotonyl-Histone H3 (Lys14) Rabbit pAb	WB, IP	4
PTM-540	Anti-Crotonyl-Histone H3 (Lys18) Mouse mAb	WB, IHC-P, ChIP	4
PTM-517RM	Anti-Crotonyl-Histone H3 (Lys18) Rabbit mAb	WB, ICC/IF	2

Cat #	Product Name	Applications	Citations
PTM-544RM	Anti-Crotonyl-histone H3 (Lys18/23) Rabbit mAb	WB, ICC/IF, IP	0
PTM-519	Anti-Crotonyl-Histone H3 (Lys23) Mouse mAb	WB, IHC-P, ChIP	4
PTM-545RM	Anti-Crotonyl-Histone H3 (Lys27) Rabbit mAb	WB, ICC/IF, ChIP	7
PTM-536RM	Anti-Crotonyl-Histone H3 (Lys36) Rabbit mAb	WB, ICC/IF	0
PTM-536	Anti-Crotonyl-Histone H3 (Lys36) Rabbit pAb	WB, ChIP	0
PTM-541	Anti-Crotonyl-Histone H4 (Ser1) Rabbit pAb	WB, ICC/IF, ChIP	1
PTM-521RM	Anti-Crotonyl-Histone H4 (Lys5) Rabbit mAb	WB	0
PTM-522RM	Anti-Crotonyl-Histone H4 (Lys8) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	0
PTM-530	Anti-Crotonyl-Histone H4 (Lys12) Mouse mAb	WB, ChIP	1
PTM-523	Anti-Crotonyl-Histone H4 (Lys12) Rabbit pAb	WB	2
PTM-524	Anti-Crotonyl-Histone H4 (Lys16) Mouse mAb	WB, IHC-P, IP	0

Glutarylation

Protein glutarylation is a reversible post-translational modification first identified in 2014, in which a **glutaryl** group (–CO–$(CH_2)_3$–COOH) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). This modification introduces a bulky, negatively charged moiety that changes the lysine side chain from a positive to a negative charge at physiological pH, thereby exerting a strong influence on protein conformation, stability, and interactions.

Glutarylation is closely linked to cellular metabolism, particularly the lysine and tryptophan degradation pathways, where glutaryl-CoA serves as the acyl donor. The modification is dynamically regulated by sirtuin 5 (SIRT5) and nutrient availability. It has been implicated in regulating mitochondrial metabolism, oxidative stress response, and chromatin dynamics.

Cat #	Product Name	Applications	Citations
PTM-1152	Anti-Glutaryllysine Mouse mAb	WB	7
PTM-1151	Anti-Glutaryllysine Rabbit pAb	WB	38
PTM-1154	Anti-Glutaryllysine Antibody Conjugated Agarose Beads	IAP	2

2-Hydroxyisobutyrylation

Protein 2-hydroxyisobutyrylation (Khib) is a dynamic and reversible post-translational modification first identified in 2014. It involves the covalent attachment of a **2-hydroxyisobutyryl** group (–CO–$C(CH_3)_2OH$) to the ε-amino group of **lysine** residues (see structure on page 10). This modification introduces a bulky, hydrophilic group that neutralizes lysine's positive charge and markedly alters the chemical properties of proteins, potentially influencing their structure, activity, and interactions.

Khib is catalyzed by histone acetyltransferases such as p300 and Tip60 and removed by histone deacetylases HDAC2 and HDAC3. It is widely distributed across species and cellular compartments, and has been detected on both histone and non-histone proteins. Functionally, lysine 2-hydroxyisobutyrylation is enriched in actively transcribed chromatin regions and plays an important role in gene expression regulation, glycolytic metabolism, and cellular stress responses.

Cat #	Product Name	Applications	Citations
PTM-802	Anti-2-Hydroxyisobutyryllysine Mouse mAb	WB	16
PTM-801	Anti-2-Hydroxyisobutyryllysine Rabbit pAb	WB	33
PTM-804	Anti-2-Hydroxyisobutyryllysine Antibody Conjugated Agarose Beads	IAP	33
PTM-820	Anti-2-Hydroxyisobutyryl-Histone H2A (Lys5) Rabbit pAb	WB	0
PTM-871	Anti-2-Hydroxyisobutyryl-Histone H2B (Lys12) Mouse mAb	WB, ChIP	0

Cat #	Product Name	Applications	Citations
PTM-831	Anti-2-Hydroxyisobutyryl-Histone H2B (Lys12) Rabbit pAb	WB	0
PTM-881	Anti-2-Hydroxyisobutyryl-Histone H3 (Lys14) Mouse mAb	WB	2
PTM-841RM	Anti-2-Hydroxyisobutyryl-Histone H3 (Lys14) Rabbit mAb	WB	1
PTM-882	Anti-2-Hydroxyisobutyryl-Histone H3 (Lys18) Mouse mAb	WB, IHC-P, ICC/IF, ChIP	1
PTM-843RM	Anti-2-Hydroxyisobutyryl-Histone H3 (Lys23) Rabbit mAb	WB, ICC/IF, ChIP	0
PTM-845	Anti-2-Hydroxyisobutyryl-Histone H3 (Lys79) Rabbit pAb	WB	1
PTM-854	Anti-2-Hydroxyisobutyryl-Histone H4 (Lys5) Mouse mAb	WB, ChIP	1
PTM-850	Anti-2-Hydroxyisobutyryl-Histone H4 (Lys5/8/12) Rabbit pAb	WB	1
PTM-805	Anti-2-Hydroxyisobutyryl-Histone H4 (Lys8) Rabbit pAb	WB, ICC/IF	7
PTM-808	Anti-2-Hydroxyisobutyryl-Histone H4 (Lys20) Rabbit pAb	WB	0

β-Hydroxybutyrylation

Lysine β-hydroxybutyrylation (Kbhb) is a reversible post-translational modification first identified in 2016, characterized by the covalent attachment of a **β-hydroxybutyryl** group (–CO–CH_2–CH(OH)–CH_3) to the ε-amino group of **lysine** residues (see structure on page 10). Kbhb is metabolically linked to the ketone body β-hydroxybutyrate, which serves as the acyl donor during nutrient deprivation or ketogenic conditions. The modification is dynamically regulated by enzymes such as p300, which functions as a lysine β-hydroxybutyryltransferase, and by histone deacetylases (HDACs) or sirtuins that remove the modification. β-hydroxybutyrylation is enriched at promoters of actively transcribed genes, representing a novel epigenetic mark that couples cellular metabolism to gene expression. As such, histone β-hydroxybutyrylation provides new insights into chromatin regulation and highlights the diverse physiological roles of β-hydroxybutyrate in conditions such as diabetes, epilepsy, and cancer.

Cat #	Product Name	Applications	Citations
PTM-1201RM	Anti-β-Hydroxybutyryllysine Rabbit mAb	WB, IHC-P, ICC/IF	23
PTM-1204	Anti-β-Hydroxybutyryllysine Antibody Conjugated Agarose Beads	IAP	12
PTM-1270	Anti-β-Hydroxybutyryl-Histone H2A (Lys5) Mouse mAb	WB	0
PTM-1220	Anti-β-Hydroxybutyryl-Histone H2A (Lys5) Rabbit pAb	WB	1
PTM-1259	Anti-β-Hydroxybutyryl-Histone H2A/H2A.X (Lys9) Rabbit pAb	WB	0
PTM-1224	Anti-β-Hydroxybutyryl-Histone H2A (Lys118) Rabbit pAb	WB	1
PTM-1225	Anti-β-Hydroxybutyryl-Histone H2A.X (Lys134) Rabbit pAb	WB	0
PTM-1230	Anti-β-Hydroxybutyryl-Histone H2B (Lys5) Rabbit pAb	WB	2
PTM-1231	Anti-β-Hydroxybutyryl-Histone H2B (Lys11) Rabbit pAb	WB	0
PTM-1232	Anti-β-Hydroxybutyryl-Histone H2B (Lys12) Rabbit pAb	WB	0
PTM-1233RM	Anti-β-Hydroxybutyryl-Histone H2B (Lys15) Rabbit mAb	WB	0
PTM-1234	Anti-β-Hydroxybutyryl-Histone H2B (Lys16) Rabbit pAb	WB	0
PTM-1235	Anti-β-Hydroxybutyryl-Histone H2B (Lys20) Rabbit pAb	WB	1
PTM-1236	Anti-β-Hydroxybutyryl-Histone H2B (Lys23) Rabbit pAb	WB	1
PTM-1238	Anti-β-Hydroxybutyryl-Histone H2B (Lys34) Rabbit pAb	WB	1
PTM-1260	Anti-β-Hydroxybutyryl-Histone H3 (Lys4) Rabbit pAb	WB, ChIP	1
PTM-1250RM	Anti-β-Hydroxybutyryl-Histone H3 (Lys9) Rabbit mAb	WB, IHC-P	5
PTM-1292	Anti-β-Hydroxybutyryl-Histone H3 (Lys18) Mouse mAb	WB	2
PTM-1293	Anti-β-Hydroxybutyryl-Histone H3 (Lys27) Mouse mAb	WB	2
PTM-1257	Anti-β-Hydroxybutyryl-Histone H3 (Lys27) Rabbit pAb	WB	0
PTM-1205	Anti-β-Hydroxybutyryl-Histone H4 (Lys5) Rabbit pAb	WB	3
PTM-1253RM	Anti-β-Hydroxybutyryl-Histone H4 (Lys8) Rabbit mAb	WB, ICC/IF	2
PTM-1206RM	Anti-β-Hydroxybutyryl-Histone H4 (Lys12) Rabbit mAb	WB, ICC/IF, ChIP	1
PTM-1206	Anti-β-Hydroxybutyryl-Histone H4 (Lys12) Rabbit pAb	WB, ChIP	5
PTM-1262	Anti-β-Hydroxybutyryl-Histone H4 (Lys16) Mouse mAb	WB, ChIP	1

Benzoylation

Lysine benzoylation (Kbz) is a recently identified post-translational modification in which a **benzoyl** group (–CO–C_6H_5) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). First reported in 2018, Kbz represents an aromatic acylation that introduces a bulky hydrophobic moiety to lysine. The benzoyl group originates from benzoyl-CoA, a central intermediate in the degradation of numerous aromatic compounds by bacteria and gut microflora, or in mammalian cells from the metabolism of the food preservative sodium benzoate. Enzymes such as ACSS2 (acyl-CoA synthetase short-chain member 2) and p300/CBP have been shown to catalyze lysine benzoylation, while SIRT2 can remove this modification, indicating its dynamic and reversible nature.

Cat #	Product Name	Applications	Citations
PTM-762	Anti-Benzoyllysine Mouse mAb	WB, IHC-P	11

L-Lactylation

Lysine L-lactylation ($K_{L\text{-}la}$) is a reversible post-translational modification first identified in 2019, involving the covalent attachment of an **L-lactyl** group (–CO–CH(OH)–CH_3) to the ε-amino group of **lysine** residues (see structure on page 10). The extent and dynamics of this modification are highly reliant on lactate levels within the cellular microenvironment and can be modulated through the introduction of extracellular lactate in cultured cells or the stimulation of intracellular glycolysis. The acetyltransferase p300 is responsible for introducing lysine L-lactylation, while Class I histone deacetylases (HDAC 1-3) have been identified as an eraser of the lactylation marks on histones. Although initially identified on histone proteins, L-lactylation has since been detected on a broad range of non-histone substrates. Among the known isomers, including D-lactylation and N-ε-carboxyethylation, L-lactylation is the predominant form found in cells and the major participant in glycolysis and the Warburg effect.

Cat #	Product Name	Applications	Citations
PTM-1401RM	Anti-L-Lactyllysine Rabbit mAb	WB, IHC-P, ICC/IF, FC, IP, ChIP	209
PTM-1404	Anti-L-Lactyllysine Antibody Conjugated Agarose Beads	IAP	52
PTM-1422RM	Anti-L-Lactyl-Histone H2A.Z (Lys11) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	3
PTM-1426RM	Anti-L-Lactyl-Histone H2B (Lys15) Rabbit mAb	WB, ChIP	1
PTM-1424RM	Anti-L-Lactyl-Histone H2B (Lys16) Rabbit mAb	WB	6
PTM-1423	Anti-L-Lactyl-Histone H2B (Lys120) Rabbit pAb	WB	1
PTM-1419RM	Anti-L-Lactyl-Histone H3 (Lys9) Rabbit mAb	WB, IHC-P, ChIP	55
PTM-1414RM	Anti-L-Lactyl-Histone H3 (Lys14) Rabbit mAb	WB, ICC/IF, ChIP	33
PTM-1406RM	Anti-L-Lactyl-Histone H3 (Lys18) Rabbit mAb	WB, IHC-P, ICC/IF, IP	103
PTM-1427RM	Anti-L-Lactyl-Histone H3 (Lys18) Rabbit mAb (ChIP Grade)	WB, ChIP, CUT&Tag	61
PTM-1413RM	Anti-L-Lactyl-Histone H3 (Lys23) Rabbit mAb	WB, IHC-P, ChIP	12
PTM-1428	Anti-L-Lactyl-Histone H3 (Lys27) Rabbit pAb	WB, ChIP	8
PTM-1421RM	Anti-L-Lactyl-Histone H3 (Lys56) Rabbit mAb	WB, FC	11
PTM-1409	Anti-L-Lactyl-Histone H4 (Lys5) Mouse mAb	WB, FC	8
PTM-1407RM	Anti-L-Lactyl-Histone H4 (Lys5) Rabbit mAb	WB, ICC/IF, FC, IP, CUT&Tag	29
PTM-1415RM	Anti-L-Lactyl-Histone H4 (Lys8) Rabbit mAb	WB, ChIP, CUT&Tag	28
PTM-1411RM	Anti-L-Lactyl-Histone H4 (Lys12) Rabbit mAb	WB, IHC-P, IP, ChIP	47
PTM-1417RM	Anti-L-Lactyl-Histone H4 (Lys16) Rabbit mAb	WB, IHC-P, ICC/IF, ChIP	18

Methacrylation

Lysine methacrylation (Kmea) is a novel post-translational modification first reported in 2021, in which a **methacryl** group (–CO–C(CH_3)=CH_2) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). Kmea is a structural isomer of crotonyllysine but exhibits a distinct mechanism and function. The metabolic precursor of Kmea is methacrylate, and the modification is dynamically regulated by enzymes: HAT1 acts as the writer, while SIRT1 and SIRT2 function as erasers. Proteomic analyses have identified 27 histone Kmea sites in HeLa cells, highlighting its widespread occurrence and potential regulatory roles.

Lysine methacrylation is metabolically linked to valine catabolism, where methacrylyl-CoA serves as the acyl donor. Accumulation of methacrylyl-CoA has been observed in patients with Leigh syndrome (LS), a neurological disease characterized by mitochondrial defects, due to mutations in short-chain enoyl-CoA hydratase (ECHS1) or 3-hydroxyisobutyryl-CoA hydrolase (HIBCH). The discovery of Kmea suggests a novel pathological mechanism linking methacrylyl-CoA accumulation to disease.

Cat #	Product Name	Applications	Citations
PTM-1501	Anti-Methacryllysine Mouse mAb	WB, IHC-P	3
PTM-1503	Anti-Methacryl-Histone H3 (Lys18) Mouse mAb	WB	2

D-Lactylation

Lysine D-lactylation ($K_{D\text{-}la}$) is a reversible post-translational modification first reported in 2024, in which a **D-lactyl** group (–CO–CH(OH)–CH_3) is covalently attached to the ε-amino group of **lysine** residues (see structure on page 10). Unlike L-lactylation, D-lactylation forms primarily through a non-enzymatic reaction between proteins and S-D-lactoylglutathione (LGSH), a byproduct of the glyoxalase pathway. In this pathway, glyoxalase 1 (GLO1) conjugates methylglyoxal (MGO), a glycolysis byproduct, with glutathione to form LGSH, which is then hydrolyzed by glyoxalase 2 (GLO2) to produce D-lactate while regenerating cellular glutathione.

D-lactate is not typically produced by human metabolism but is mainly generated by microorganisms, including certain bacteria and yeasts, during fermentation. Accumulation of D-lactate under conditions such as intestinal microbiota dysbiosis, short bowel syndrome, or specific disease states can lead to D-lactic acidosis, characterized by low blood bicarbonate, decreased pH, hyperuricemia, and in severe cases, neurological symptoms such as seizures, ataxia, and altered consciousness.

Cat #	Product Name	Applications	Citations
PTM-1429RM	Anti-D-Lactyllysine Rabbit mAb	WB, ICC/IF, IP	4
PTM-1434	Anti-D-Lactyllysine Antibody Conjugated Agarose Beads	IAP	0

About PTM BIO

Founded in 2011, PTM BIO is a global leader in post-translational modification research, combining scientific innovation, production, and technical expertise. For 15 years, PTM BIO has pioneered the development of exclusive PTM antibodies and IVD raw materials, advanced proteomics solutions, and integrated bioinformatics tools that accelerate biomedical and pharmaceutical research. Driven by innovation and excellence, PTM BIO is dedicated to continuously shaping the future of precision medicine.

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