
Epigenetic
Modifiers
DNA Methyltransferase
Histone Deacetylase
Histone Methyltransferase
Aurora Kinase
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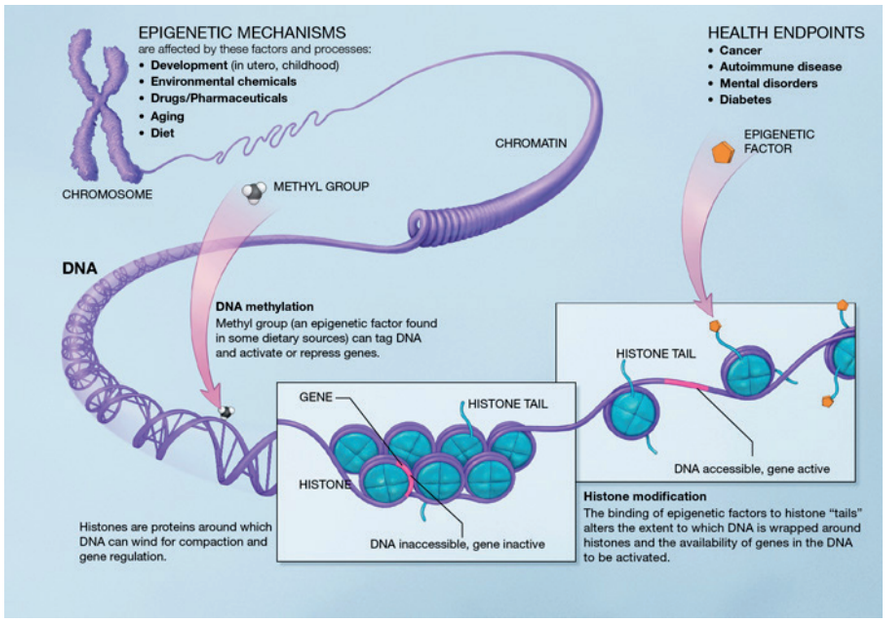
Biochemicals for Life Science Research

Introduction to Epigenetics

Epigenetics involves changes in gene activity that are not directly caused by DNA sequence alterations. Instead, gene expression is activated or inhibited through regulatory changes such as chromatin remodeling, which can involve modifications of histone proteins or the addition of methyl or alkyl groups to nucleotides.

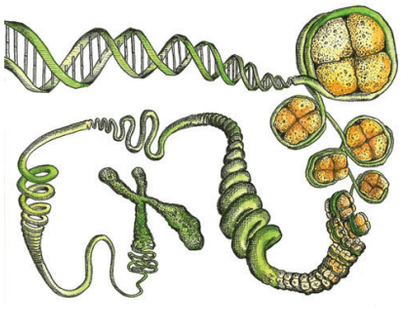
Methylation or acetylation of histones can have profound changes on the state of chromatin and its probability of transcription. Similarly, hypermethylation of DNA sequences in the promoter region of a gene can prevent transcription of the gene product. Targeting modifiers like deacetylases and methyltransferases shows potential for therapeutic benefit in the treatment of a variety of cancers.



DNA Methyltransferase (DNMTs)

DNA methyltransferases (DNMTs) predominantly methylate CpG nucleotide sequences on DNA. Three active DNA methyltransferases have been identified in mammals: DNMT1, DNMT3A, and DNMT3B. Typically, methylation of these sequences in promoter regions prevents expression of genes by physically preventing transcription factors from binding to DNA. Additionally, methylated DNA can be bound by methyl-CpG-binding domain proteins that recruit histone remodeling enzymes; these enzymes can condense chromatin structure, offering another mechanism for suppressing gene expression. In some cancers, this results in decreased expression of tumor suppressor genes and unregulated cell growth. Several compounds inhibit activity of DNA methyltransferase, including RG-108, mithramycin, and azacytidine.

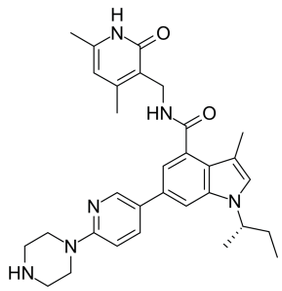
Cat #	Product Name	Description	Purity
A9602	Azacitidine	Inhibits DNMT1/3A/3B	≥98%
A9603	5-Aza-2'-deoxycytidine (Decitabine)	Inhibits DNMT1/3A/3B	≥98%
B3573	Bisdemethoxycurcumin	Inhibits DNMT1	≥98%
C2944	Chlorogenic Acid	Inhibits DNMT	≥98%
E6234	Epigallocatechin Gallate	Inhibits DNMT1	≥98%
F3473	Fisetin	Inhibits DNMT1	≥97%
L5750	Lomeguatrib	Inhibits O6-methylguanine-DNMT (MGMT)	≥98%
M3476	Mithramycin	Inhibits DNMT1	≥98%
B1855	O6-Benzylguanine	Inhibits O6-methylguanine-DNMT (MGMT)	≥98%
R2400	RG-108	Inhibits DNMT	≥98%
S3352	Sinefungin	Inhibits DNMT	≥95%
S5868	Sorafenib	Inhibits DNMT activity	≥98%
T286163	Theaflavin-3,3'-digallate	Inhibits DNMT	≥98%



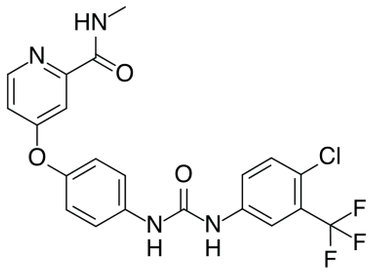
Histone Methyltransferase Inhibitors

Cat #	Product Name	Description	Purity
E6396	EPZ005687	Inhibits EZH2	≥99%
E6398	EPZ5676	Inhibits DOT1L	≥98%
E6397	EPZ6438	Inhibits EZH2	≥99%
G7340	GSK126	Inhibits EZH2	≥99%
G7442	GSK343	Inhibits EZH2	≥98%
S5868	Sorafenib	Inhibits EZH2	≥98%

Histone methyltransferases transfer methyl groups to lysine and arginine residues of histones, particularly on histones H3 and H4. Methylation of histones makes them more neutral in charge, allowing them to separate slightly from DNA; this loose conformation makes the DNA more easily accessible. Histone methyltransferases can activate gene expression in this manner, as transcription of DNA sequences more loosely wrapped around methylated histones is more likely to occur. However, depending on the histone, this same process can also silence gene transcription, as methylation may block the DNA binding and activation sites for some transcription factors or induce chromatin condensation. In some forms of cancer, methylation of histones by methyltransferases EZH2 or DOT1L silences expression of tumor suppressor genes. Inhibitors of histone methyltransferases such as EPZ5676, EPZ005687, and GSK126 exhibit anticancer chemotherapeutic activity across a variety of in vitro and in vivo cancer models.



G7340 GSK126



S5868 Sorafenib

Histone Deacetylases (HDACs)

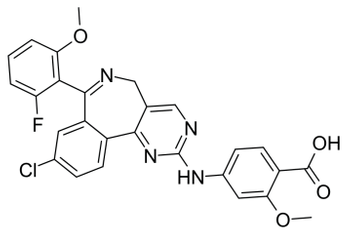
Cat #	Product Name	Description	Purity
A4002	AK-7	Inhibits SIRT2 (HDAC class III), brain penetrant	≥98%
A6132	Apicidin	Inhibits HDAC (broad spectrum, class I/II)	≥98%
B1746	Belinostat	Inhibits HDAC	≥98%
B8276	Butyric Acid Sodium	Inhibits HDAC	≥97%
C0048	Cambinol	Inhibits SIRT1 (HDAC class III)	≥98%
C8069	Curcumin	Decreases expression of HDAC3 (class I)	≥98%
E5477	Entinostat	Inhibits HDAC1 (class I)	≥98%
I7559	Isoliquiritigenin	Inhibits HDAC (class I/IIA)	≥98%
L0528	LBH-589	Inhibits HDAC1/2/3/11 (class I)	≥98%
M2409	MGCD-0103	Inhibits HDAC	≥98%
M9710	Mycophenolic Acid	Inhibits HDAC	≥98%
P2815	Phenylbutyrate	Inhibits HDAC	≥98%
R5749	Romidepsin	Inhibits HDAC	≥98%
S0344	Salermide	Inhibits SIRT1/2 (HDAC class III)	≥98%
S3470	Sirtinol	Inhibits SIRT1/2 (HDAC class III)	≥98%
S1069	Scriptaid	Inhibits HDAC (broad spectrum)	≥98%
S5868	Sorafenib	Decreases expression of HDAC1/2/4/5/8 (classI/IIA)	≥98%
T5060	TMP-269	Inhibits HDAC (class II)	≥98%
T5996	Tozasertib	Decreases expression of HDAC	≥98%
T6933	Trichostatin A	Inhibits HDAC1/3/4/6/10 (class I/IIA/IIB)	≥98%
T8000	Tubacin	Inhibits HDAC6/10 (class IIB)	≥98%
T8006	Tubastatin A HCl	Inhibits HDAC6/10 (class IIb)	≥98%
V0144	n-Valeric Acid	Inhibits HDAC	≥98%
V0147	Valproic Acid Na^+ Salt	Inhibits HDAC1 (class I)	≥98%
V5734	Vorinostat (SAHA)	Inhibits HDAC1/2/3/6 (class I/IIB)	≥98%

Histone deacetylases (HDACs) are responsible for removing acetyl groups from N-acetyl lysine amino acids on histones, making them more positively charged and able to more tightly bind the negatively charged DNA backbone. As a result, DNA structure condenses and genetic transcription is less likely to occur. HDACs can prevent expression of genes important in apoptosis and tumor suppression. HDACs are subdivided into four separate groups based on their localization and function. Class I HDACs (isotypes 1, 2, 3, 8) are primarily found in the nucleus, whereas class II HDACs (isotypes 4, 5, 6, 7, 9, 10) are able to travel through the nuclear membrane and are found in both the nucleus and the cytoplasm. HDAC inhibitors exhibit anticancer activity when co-administered with other chemotherapeutics, particularly in the treatment of leukemias and lymphomas. HDAC inhibitors include vorinostat, trichostatin A, scriptaid, and phenylbutyrate.

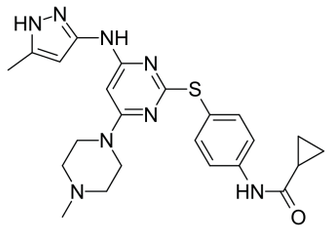
Aurora Kinase

The aurora kinases are a family of proteins that regulate mitosis. Three kinases, Aurora A, Aurora B, and Aurora C, are each responsible for different functions involving chromatid segregation and other mechanisms of cellular division. They are overexpressed in a variety of cancers, making them interesting therapeutic targets for cancer research. Inhibition of each of the aurora kinases induces apoptosis through unique mechanisms. Aurora A inhibition disrupts mitotic spindle assembly, while Aurora B interferes with chromosome alignment. The role of Aurora C is less known as it is typically expressed in meiotic cells, however it has recently been shown to demonstrate oncogenic activity. Aurora A and B have thus been the focus of small molecule targeting to date.

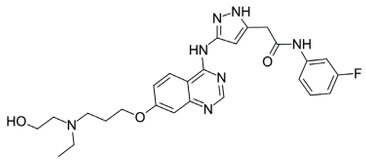
ID	Name	Description	Purity
A9714	AZD-1152-HQPA (Barasertib)	Determined to be the most selective available AurB inhibitor in a 2015 study.	≥98%
C9708	CYC-116	Inhibits AurA and AurB. Induces apoptosis in multiple myeloma cells in combination with matrine.	≥98%
G7444	GSK-1070916	Inhibits AurB and AurC.	≥98%
M4652	MLN8237 (Alisertib)	Selective AurA inhibitor. Effective in treating models of neruoblastoma, acute lymphoblastic leukemia, and sarcoma.	≥98%
T5996	VX680 (Tozasertib)	AurA inhibitor with some AurB inhibitory effect.	≥98%
Z4900	ZM-447439 Trihydrate	Inhibits AurA and AurB. Limits migration of MCF-7 human breast cancer cells.	≥98%



M4652 MLN8237



T5996 Tozasertib



A9714 AZD-1152-HQPA

About Us

LKT Labs is focused on the production and distribution of high purity biochemicals for all types of life science research, including cancer biology, immunology, cardiovascular studies, microbiology, and neuroscience. Our product library includes a wide variety of compounds that can be used for a broad spectrum of in vitro and in vivo research applications.

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