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-Product purification
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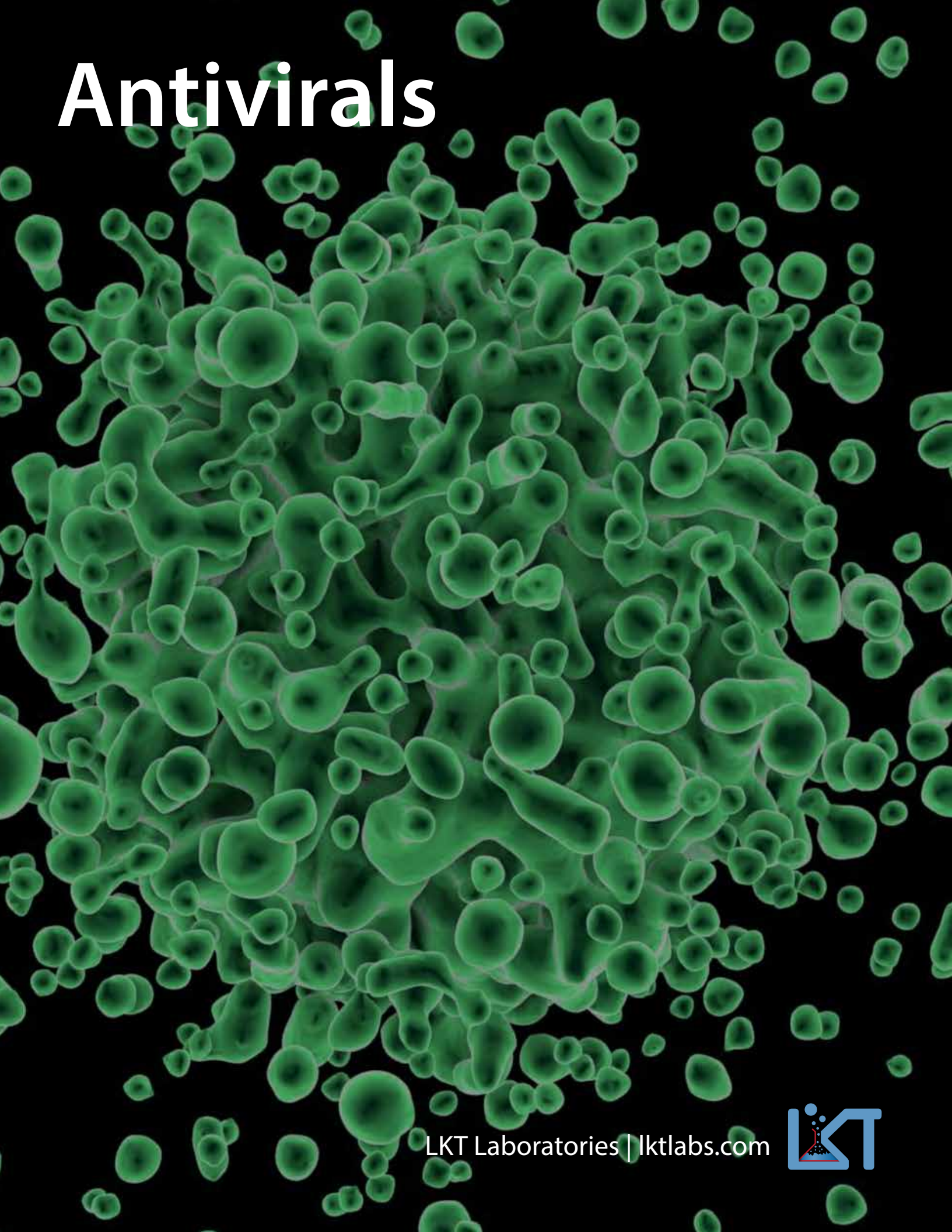
Antivirals



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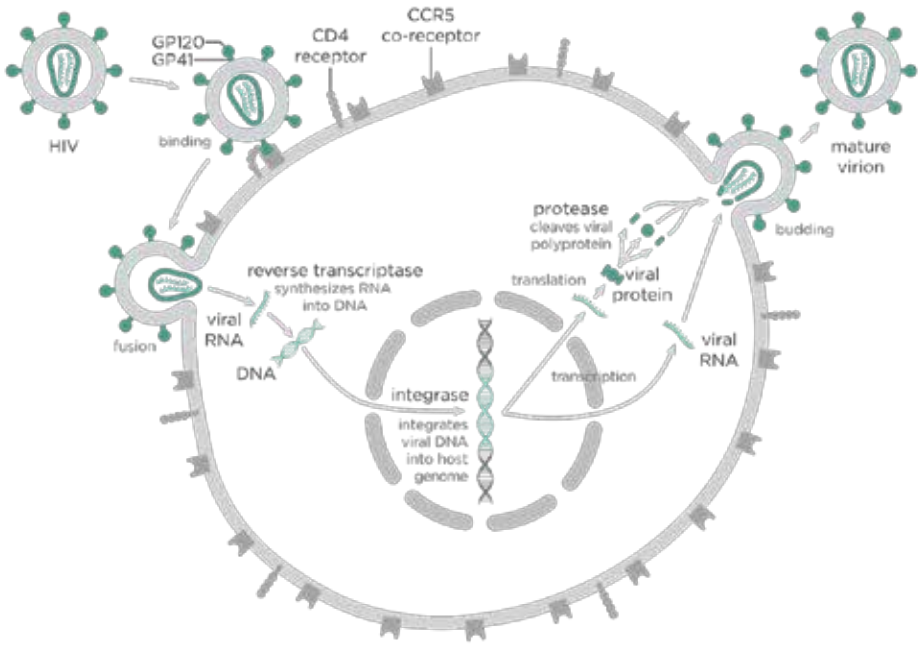
Introduction to Antivirals

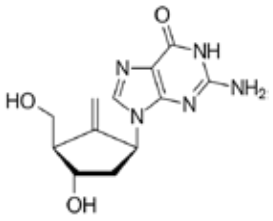
Viruses are infectious agents that use host cell machinery to replicate rather than their own. The life cycle of a virus includes several stages: attachment, entry, replication, assembly, and release. During the attachment stage, viral capsid proteins bind host cell surface receptors. After this binding, the viral and cell membranes may fuse, allowing viral entry into the cell. Once in the cell, viral DNA is produced and integrated into host cell DNA, allowing the host cell to begin replicating viral DNA. This viral DNA is processed and translated into proteins that are assembled into virus particles such as viral capsids or envelopes. Viruses are released from host cells either through budding, an exocytosis-like process, or cell lysis.



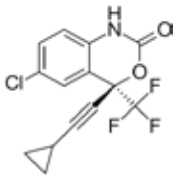
Reverse Transcriptase Inhibitors

Cat #	Product Name	Description	Purity
A0401	Abacavir	Inhibits HIV reverse transcriptase	≥98%
A0402	Abacavir Sulfate	Inhibits HIV reverse transcriptase	≥98%
D0261	Dapivirine	Inhibits HIV reverse transcriptase	≥98%
D3212	2',3'-Dideoxycytidine	Inhibits HIV reverse transcriptase	≥98%
D3214	2',3'-Dideoxyinosine	Inhibits HIV reverse transcriptase	≥98%
E2003	Efavirenz	Inhibits HIV reverse transcriptase	≥98%
E5178	Emtricitabine	Inhibits HIV and HBV reverse transcriptase	≥98%
E5576	Entecavir	Inhibits HIV and HBV reverse transcriptase	≥97%
G0243	(-)-Gallocatechin	Inhibits HIV integrase and reverse transcriptase	≥98%
L0350	Lamivudine	Inhibits HIV and HBV reverse transcriptase	≥98%
Q8016	Quercetin Dihydrate	Inhibits MMLV and RAV-2 reverse transcriptase	≥95%
R8207	β-Rubromycin	Inhibits HIV reverse transcriptase	≥98%
S7603	Stavudine	Inhibits HIV reverse transcriptase	≥98%
T1854	Tenofovir Monohydrate	Inhibits HIV and HBV reverse transcriptase	≥98%

Reverse transcriptase (RT) is an enzyme used to generate complimentary DNA (cDNA) from RNA during reverse transcription. This enzyme complex includes an RNA-dependent DNA polymerase and a DNA-dependent DNA polymerase that work together to perform transcription. cDNA encoded by RTs is incorporated into the host genome and replicated during the spread of infection. RTs are primarily associated with retroviruses but can also be found in other viruses, including hepatitis B virus.



E5576 Entecavir

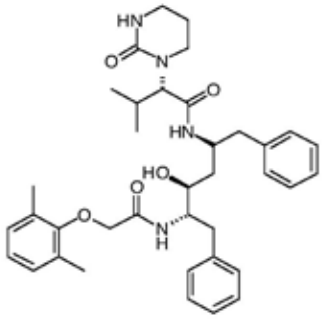


E2003 Efavirenz

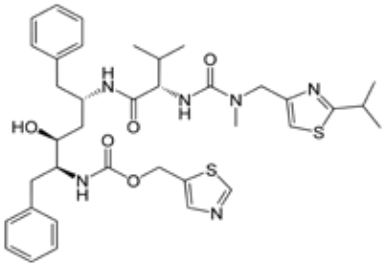
Protease Inhibitors

Viral proteases are enzymes involved in proteolytic cleavage of protein precursors necessary for production of infection viral particles such as the viral capsid or envelope proteins required to create a mature virion. HIV-1 protease is a retroviral aspartyl protease; without activity of this protease, virions are not infectious. Inhibition of proteases prevents the virus from being able to infect other cells.

Cat #	Product Name	Description	Purity
D1872	Des(benzylpyridyl) Atazanavir	Inhibits HIV protease	≥98%
G3353	Ginkgolic Acid	Inhibits HIV protease	≥98%
G3352	Ginkgolic Acid (13:0)	Inhibits HIV protease	≥98%
G3351	Ginkgolic Acid Mixture	Inhibits HIV protease	≥98%
I5313	Indinavir Sulfate	Inhibits HIV protease, GLUT4, and calpain	≥98%
L5862	Lopinavir	Inhibits HIV protease and SERCA	≥98%
N5550	Nomilin	Inhibits HIV protease	≥98%
R3577	Ritonavir	Inhibits HIV protease	≥98%
V9200	VX-950	Inhibits HCV NS3/4A protease	≥98%



L5862 Lopinavir

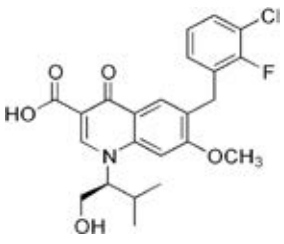


R3577 Ritonavir

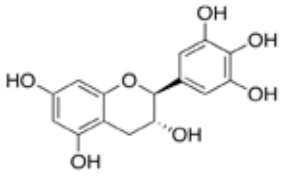
Integrase Inhibitors

Cat #	Product Name	Description	Purity
H3275	HCKFWW Peptide	Inhibits HIV integrase	≥95%
C0253	Candesartan	Inhibits interaction between LEDGF and HIV-1 integrase	≥98%
C0254	Candesartan Celexetil Ester	Inhibits interaction between LEDGF and HIV-1 integrase	≥98%
G0245	Gallocatechin Gallate	Inhibits HIV integrase	≥98%
G0243	(-)-Gallocatechin	Inhibits HIV integrase and reverse transcriptase	≥98%
E4785	Elvitegravir	Inhibits HIV integrase	≥98%
R0247	Raltegravir	Inhibits HIV integrase	≥98%

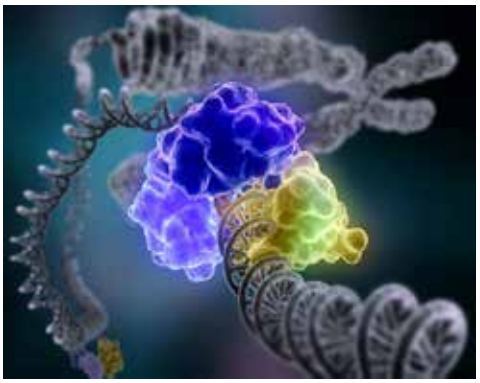
Integrase is an enzyme produced by retroviruses that enables viral genetic matter to be incorporated into the DNA of the host cell. Integrase takes the DNA produced by reverse transcriptase and catalyzes 3' processing and strand transfer to initiate ligation of viral DNA to host DNA. After this step, the host cell becomes a permanent carrier for the virus as it is often unable to differentiate between host and viral DNA.



E4785 Elvitegravir



G0243 (-)-Gallocatechin



Integrase prepares viral DNA for ligation

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Epigenetic modifiers

Histone methyltransferase inhibitors

DNA methyltransferase inhibitors

Histone deacetylase inhibitors

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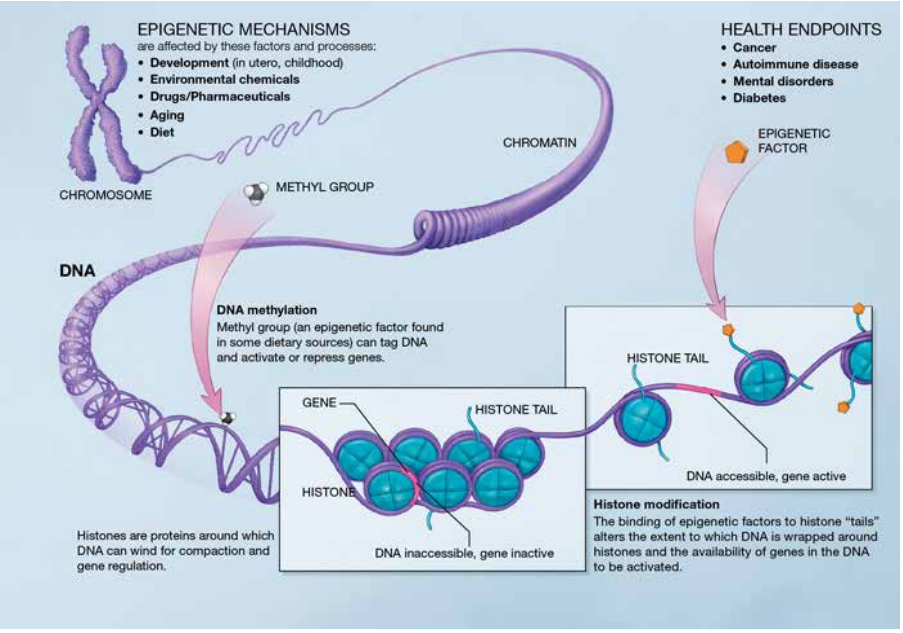
Introduction to Epigenetics

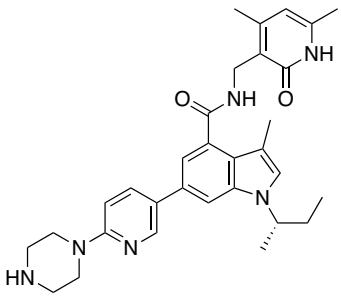
Epigenetics involves changes in gene activity that are not directly caused by DNA sequence alterations. Instead, gene expression is activated or inhibited through regulatory changes such as chromatin remodeling, which can involve modifications of histone proteins or the addition of methyl or alkyl groups to nucleotides. Methylation or acetylation of histones can have profound changes on the state of chromatin and its probability of transcription. Similarly, hyper-methylation 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.



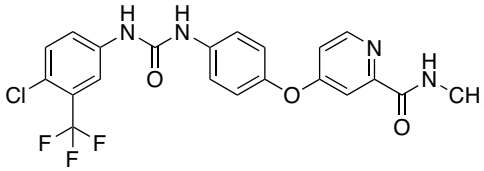
Histone Methyltransferase Inhibitors

Cat #	Product Name	Description	Purity
E6298	EPZ004777	Inhibits DOT1L	≥98%
E6396	EPZ005687	Inhibits EZH2	≥99%
E6398	EPZ5676	Inhibits DOT1L	≥98%
E6397	EPZ6438	Inhibits EZH2	≥99%
G7340	GSK126	Inhibits EZH2	≥99% ee
G7442	GSK343	Inhibits EZH2	≥98%
S5868	Sorafenib	Inhibits EZH2	≥98%
S5869	Sorafenib Tosylate	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

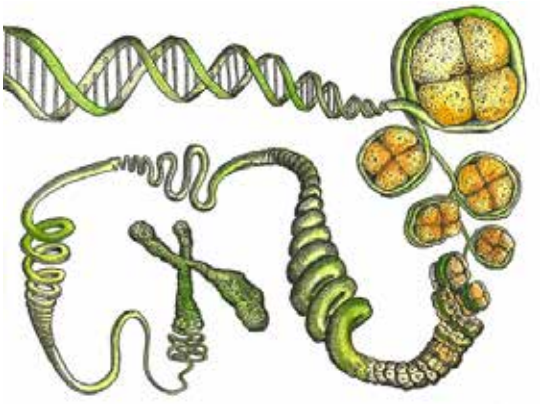


S5868 Sorafenib

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%
B3573	Bisdemethoxycurcumin	Inhibits DNMT1	≥98%
C2944	Chlorogenic Acid	Inhibits DNMT	≥98%
A9603	Decitabine	Inhibits DNMT1/3A/3B	≥98%
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%
S5868	Sorafenib	Inhibits DNMT activity	≥98%
S5869	Sorafenib Tosylate	Inhibits DNMT activity	≥98%



Histone Deacetylases (HDACs)

Cat #	Product Name	Description	Purity
A6132	Apicidin	Inhibits HDAC (broad spectrum, class I/II)	≥98%
B1746	Belinostat	Inhibits HDAC	≥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%
S1069	Scriptaid	Inhibits HDAC (broad spectrum)	≥98%
B8276	Sodium Butyrate (Butyric Acid)	Inhibits HDAC	≥97%
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.

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LKT Laboratories

Neuroscience Research Chemicals

Glioma Chemotherapeutics | Plant- and Animal-based Products
Neurotransmitter Modulators | Ion Channel Modulators

Neuroscience Research Chemicals

Neuroscience is a widely interdisciplinary field and centrally-mediated signaling plays an important role in a variety of diseases and dysfunctions, such as cancer, depression, and epilepsy. Our product library includes an assortment of neuroscience-focused compounds necessary to study such disorders, including glioma chemotherapeutics, neurotransmitter modulators, plant- and animal-based products, and ion channel modulators, among many others

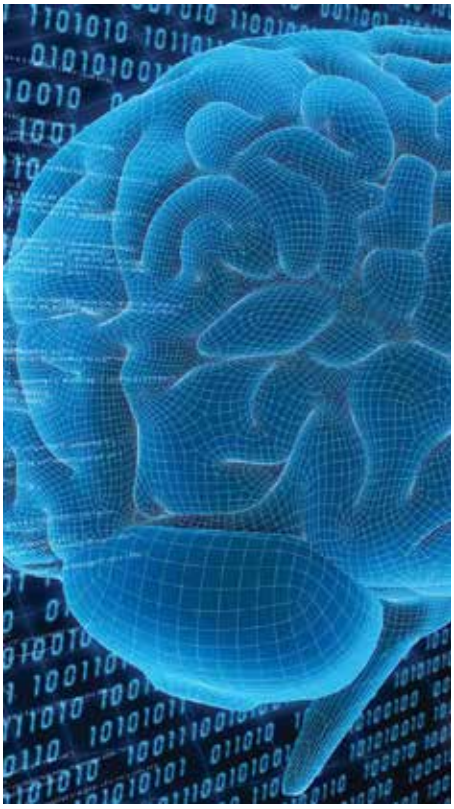
Glioma chemotherapeutics
Glioma chemotherapeutics show pre-clinical and clinical benefit in the treatment of centrally-located or cranial cancers such as oligodendrocytoma and glioblastoma multiforme. These compounds act on a wide variety of targets, such as receptor tyrosine kinases, serine/threonine kinases, tubulin, DNA replication enzymes, and DNA itself. Products acting on DNA itself include intercalators and alkylating agents such as temozolomide (pg. 3), which inhibits DNA synthesis and replication. There are also a wide variety of enzyme inhibitors, such as MK-2206 (pg. 3), O^6-benzylguanine (pg. 2), and vorinostat (pg. 3), which respectively inhibit Akt, O^6-methylguanine DNA methyltransferase, and histone deacetylase.

Neurotransmitter modulators
Neurotransmitter modulators include compounds that alter neurotransmitter levels through action on neurotransmitters themselves, receptors, enzymes, and other related proteins. Many compounds that we carry act directly on receptors, such as baclofen (pg. 4), an agonist at $GABA_B$ receptors, and mirtazapine (pg. 5), an antagonist at 5-HT receptors and α2-adrenergic receptors. Other compounds inhibit neurotransmitter reuptake, such as fluoxetine (pg. 5), a 5-HT transporter inhibitor, and many products have multiple mechanisms of action on multiple neurotransmitters, such as bupropion (pg. 5), which acts on DA and NE transporters as well as nAChRs.

Plant- and animal-based products
Compounds that can be found in nature have been used traditionally for many years and often offer a great variety of medicinal benefits; these typically have very distinct mechanisms of action that include a multitude of targets as well, ranging from antioxidative transcription factors to neurotransmitter-degrading enzymes. Resveratrol (pg. 6) is one of many products known for its antioxidative capacities, activation of SIRT1, and modulation of MAO. Berberine (pg. 6) is an inhibitor of AChE and prolyl oligopeptidase found in the barberry plant, the California poppy, and the Amur cork tree. Additionally, kawain (pg. 6) is one of many lactones found in the roots of the kava plant, which activates Nrf2 and modulates signaling of Na^+, K^+, and Ca^{2+} ion channels.

Ion channel modulators
Ion channel modulators alter ion channel signaling and are often used as antiepileptics/anticonvulsants, analgesics, and anesthetics. Some compounds directly alter signaling of one specific subtype of channel, such as flupirtine (pg. 7), which activates KCNQ/K_v7 K+ channels, and bulleyaconitine (pg. 7), which inhibits voltage-gated Na^+ channels. Others directly modulate signaling of many ion channels, such as oxcarbazapine (pg. 7), which inhibits voltage-gated Na^+ and K^+ channels. Additional compounds act indirectly, targeting upstream or downstream signaling proteins, such as levetiracetam (pg. 7), which inhibits Ca^{2+} signaling by binding to synaptic vesicle glycoprotein SV2A.

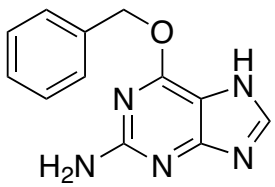
Glioma Chemotherapeutics



O^6-benzylguanine

O^6-benzylguanine (O6-BG) is a synthetic guanine derivative often used to study DNA repair mechanisms. O6-BG is an inhibitor of O6-methylguanine DNA methyltransferase, an enzyme that repairs damage to guanine residues in DNA. As many alkylating and cross-linking chemotherapeutics act on guanine residues to induce DNA damage in cancer cells, O6-BG prevents repair of the damaged DNA, allowing apoptosis and other mechanisms of cell death to occur[1]. In animals and humans, O6-BG shows some benefit in improving efficacy of co-administered treatments, potentially increasing survival time[2,3,4].

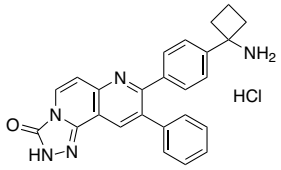
1. Quinn JA, et al. Clin Cancer Res. 2009 Feb 1;15(3):1064-8.
2. Quinn JA, et al. J Clin Oncol. 2009 Mar 10;27(8):1262-7.
3. Qian L, et al. Biomaterials. 2013 Nov;34(35):8968-78.
4. Friedman HS. Clin Cancer Res. 2000 Aug;6(8):2967-8.



Glioma Chemotherapeutics

Cat #	Product Name	Description	Purity
B1855	O^6-Benzylguanine	Inhibits MGMT	≥98%
B5871	Bortezomib	Inhibits 26S proteasome	≥98%
C0171	Carboplatin	Guanine cross-linking agent	≥98%
C0173	Carmustine	Alkylating agent	≥98%
C3374	Cisplatin	Guanine cross-linking agent	≥98%
C9609	Cyclophosphamide	Inhibits T_{reg} proliferation; alkylating agent	≥98%
D0375	Dasatinib Monohydrate	Inhibits EphA/B, BCR-abl, c-kit, src	≥98%
E6846	Erlotinib MonoHCL	Inhibits EGFR	≥98%
E7657	Etoposide	Inhibits Topo II	≥98%
E8419	Everolimus	Inhibits mTORC1	≥98%
G1721	Gefitinib	Inhibits EGFR	≥98%
I2056	Ifosfamide	Alkylating agent	≥98%
I4802	Imatinib Mesylate	Inhibits Abl, c-Kit, PDGFR	≥98%
I6932	Irinotecan	Inhibits Topo I	≥98%
L0360	Lapatinib Ditosylate	Inhibits EGFR (HER2)	≥97%
L5648	Lomustine	Alkylating agent	≥98%
M1676	Methotrexate hydrate	Inhibits DHFR (purine synthesis), IL-1R, CLA	≥98%
M3379	Mitoxantrone DiHCL	Inhibits Topo II; intercalating agent	≥98%
M4000	MK-2206	Inhibits Akt	≥99%
P6858	Procarbazine HCL	Inhibits MAO; increases H_2O_2	≥97%
R0161	Rapamycin (Sirolimus)	Inhibits mTORC1	≥98%
S5868	Sorafenib	Inhibits VEGFR, PDGFR, C-Raf, B-Raf	≥98%
T0008	Tacrolimus	Inhibits calcineurin	≥98%
T1849	Temozolomide	Dacarbazine derivative; alkylating agent	≥98%
V5254	Vincristine Sulfate	Inhibits microtubule assembly (tubulin)	≥82%
V3251	Vinorelbine Base	Inhibits microtubule assembly (tubulin)	≥90%
V5734	Vorinostat	Inhibits HDAC; chelates Zn^{2+}	≥98%

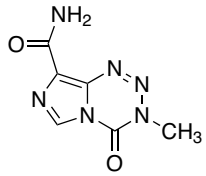
MK-2206



MK-2206 is an orally bioavailable allosteric inhibitor of Akt, preventing its phosphorylation and translocation to the cellular membrane[1]. In vitro, this compound induces cell cycle arrest and inhibits cellular proliferation in a variety of cancer cell lines[2,3]. In models of glioma, MK-2206 shows preliminary efficacy when combined with other synergistic treatments, inhibiting cell proliferation, migration, and invasion and inducing autophagy[4,5,6]. This compound is currently in phase I and II clinical trials as a treatment for a wide variety of cancers.

1. Davies BR, et al. Mol Cancer Ther. 2012 Apr;11(4):873-87.
2. Jiao P, et al. Mol Cell Biochem. 2013 Jun 25. [Epub ahead of print]
3. Burke JF, et al. Ann Surg Oncol. 2013 Jul 31. [Epub ahead of print]
4. Jin R, et al. Neurosci Lett. 2013 Feb 8;534:316-21.
5. Quayle SN, et al. PLoS One. 2012;7(11):e49466.
6. Cheng Y, et al. Mol Cancer Ther. 2012 Jan;11(1):154-64.

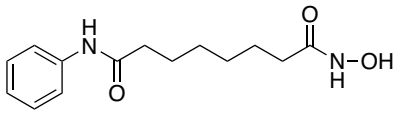
Temozolomide



Temozolomide is a second generation imidazotetrazine clinically approved to treat gliobastoma multiforme, anaplastic astrocytoma, and oligodendrocytoma[1]. Temozolomide acts as an alkylating agent, attaching alkyl groups to guanine bases and interfering with DNA replication[2]. This compound is unique as it is 100% orally bioavailable and enters the cerebrospinal fluid easily and quickly[3].

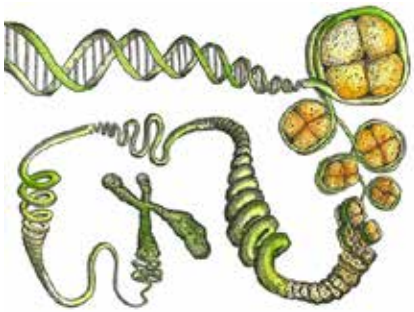
1. Nagasawa DT, et al. Neurosurg Clin N Am. 2012 Apr;23(2):307-22, ix.
2. Wesolowski JR, et al. AJNR Am J Neuroradiol. 2010 Sep;31(8):1383-4.
3. Friedman HS, et al. Clin Cancer Res. 2000 Jul;6(7):2585-97.

Vorinostat



Vorinostat (suberoylanilide hydroxamicacid) is a HDAC inhibitor that prevents deacetylation of histones, altering chromatin structure and inhibiting gene expression. In vitro, vorinostat promotes cell cycle arrest, induces apoptosis, and inhibits cellular proliferation[1,2]. This compound is very effective when co-administered with other treatments in glioblastoma stem-like cells and is currently being studied as a potential treatment for various forms of glioma[3,4]. Additionally, vorinostat attenuates impairment of fear extinction in animal models and disrupts HIV latency in HIV-infected patients, suggesting it has additional benefit beyond its anticancer activity[5,6].

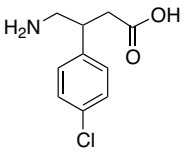
1. Silva G, et al. PLoS One. 2013;8(1):e53766.
2. Xu J, et al. J Neurooncol. 2011 Nov;105(2):241-51.
3. Asklund T, et al. Anticancer Res. 2012 Jul;32(7):2407-13.
4. Lee EQ, et al. Clin Cancer Res. 2012 Nov 1;18(21):6032-9.
5. Matsumoto Y, et al. Psychopharmacology (Berl). 2013 Sep;229(1):51-62.
6. Archin NM, et al. Nature. 2012 Jul 25;487(7408):482-5.



Neurotransmitter Modulators

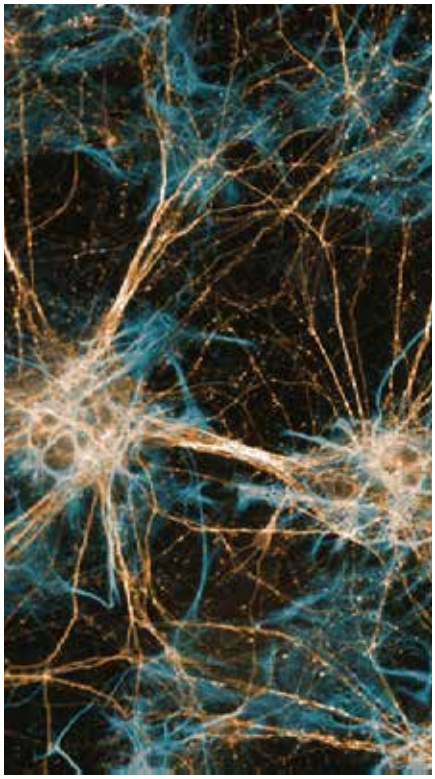
Cat #	Product Name	Description	Purity
A4802	Amantadine HCL	Inhibits α_7 nAChR, NMDAR, MAOB, viral protein M2; potentiates DA release	≥96%
A5234	Amisulpride	Inhibits $D_{2/3}R$, 5-$HT_{2B/7}R$; activates GHBR	≥98%
A5235	Amitriptyline HCL	Inhibits SERT, NET, $D_{1/3/5}R$, 5-$HT_{2/3/6/7}R$, $H_{1/4}R$, $\alpha_{1/2}R$, $M_{1\text{-}5}R$, voltage-gated Na^+, L-type Ca^{2+}, $K_V1.1/7.2/7.3$ channels; activates σ_1R, TrkA/BR	≥98%
A5059	Amoxapine	Inhibits SERT, NET, $D_{2\text{-}4}R$, 5-$HT_{2/3/6/7}R$, H_1R	≥98%
A5326	Aniracetam	Inhibits $D_{2/3}R$, 5-$HT_{2C}R$, nAChR; pot. AMPAR	≥98%
A7085	Arvanil	Activates CB_1R, TRPV1	≥98%
B0110	Baclofen	Activates $GABA_BR$	≥98%
B8363	Bupropion HCL	Inhibits NET, DAT, $\alpha_3\beta_2/\alpha_3\beta_4/\alpha_4\beta_2$ nAChR, $\alpha_{1/2}R$.	≥98%
B8274	Buspirone HCL	Inhibits $D_{2\text{-}4}R$; activates α_1R; partial ag. at 5-$HT_{1A}R$	≥98%
C0221	Caffeine	Inhibits $Ad_{1/2}R$, PDE; neg. modulates GABA	≥98%
C3472	Cisatracurium Besylate	Inhibits nAChR	≥95%
C4757	Clozapine	Inhibits $D_{1\text{-}4}R$, 5-$HT_{1/2/3/6/7}R$, $H_{1/4}R$, $\alpha_{1/2}R$, $M_{1\text{-}5}R$; activates $GABA_BR$; partial ag. at 5-$HT_{1C}R$; pot. NMDAR	≥97%
C9779	Cytisine	Activates nAChR	≥98%
D1644	Deltorphin I	Activates δOR	≥98%
D1769	Dermorphin	Activates μOR	≥96%
D1792	Dextromethorphan HBr Hydrate	Inhibits SERT, NET, $\alpha_7/\alpha_3\beta_4/\alpha_4\beta_2$ nAChR, MR, NMDAR, NADPH oxidase; activates σ_1R; pot. μOR	≥98%
D5753	Donepezil HCL	Inhibits AChE	≥98%
D5994	Doxepin HCL	Inhibits SERT, NET, 5-$HT_{1/2}R$, $H_{1/2}R$, α_1R, $M_{1\text{-}5}R$	≥98%
E5575	Entacapone	Inhibits COMT	≥98%
F4780	Fluoxetine HCL	Inhibits SERT, 5-$HT_{2A/2C}R$; activates σ_1R	≥98%
F4783	Fluvoxamine Maleate	Inhibits SERT; activates σ_1R	≥97%
G0048	GABA	Neurotransmitter; activates GABAR	≥98%
G0246	Galantamine HBr	Inhibits AChE; pot. nAChR	≥98%
H0142	Haloperidol	Inhibits $D_{1\text{-}5}R$, 5-$HT_{2A/7}R$, $\alpha_{1/2}R$, NMDAR, σ_1R; activates σ_2R	≥95%
H9714	L-5-Hydroxytryptophan	Precursor of 5-HT and melatonin	≥98%
K1678	Ketanserin	Inhibits $D_{1,2}R$, 5-$HT_{2A/2C/6}R$, H_1R, α_1R	≥97%
L1782	Levodopa	Precursor of catecholamines DA, NE, EPI	≥98%
M1708	Mecamylamine HCL	Inhibits nAChR	≥98%
M1745	Melatonin	Activates $MT_{1/2}R$	≥98%
M1749	Memantine HCL	Inhibits D_2R, 5-HT_3R, α_7 nAChR, NMDAR	≥98%
M3368	Mirtazapine	Inhibits 5-$HT_{2/3/6/7}R$, $\alpha_{1/2}R$, MR; activates 5-$HT_{1A}R$	≥98%
N1721	Nefiracetam	Activates $\alpha_3\beta_2/\alpha_3\beta_4/\alpha_4\beta_2/\alpha_4\beta_4/\alpha_7$ nAChR, PKC; pot. NMDAR	≥98%
P0252	Pancuronium Bromide	Inhibits nAChR	≥98%
P6901	Pramipexole DiHCL	Activates $D_{2\text{-}4}R$	≥98%
R0348	Ramelteon	Activates $MT_{1/2}R$	≥98%
R0272	Rasagiline	Inhibits MAOB, voltage-gated anion channels	≥98%
R3586	Rivastigmine Hydrogen Tartrate	Inhibits AChE, BChE	≥98%
S1059	Scopolamine HBr	Inhibits $M_{1\text{-}5}R$	≥98%
S1971	Sertraline HCL	Inhibits SERT, DAT, α_1R; activates σ_1R	≥98%
T2936	Thioridazine HCL	Inhibits $D_{1/2}R$, 5-$HT_{2A}R$, α_1R, voltage-gated hERG K^+ channels	≥98%
T6802	Tramadol HCL	Inhibits SERT, NET, 5-$HT_{2C}R$, α7 nAChR, $M_{1/3}R$, NMDAR; activates μOR, TRPV1	≥98%
V1854	Venlafaxine HCL	Inhibits SERT, NET, DAT	≥98%
Z5745	Zolmitriptan	Activates 5-$HT_{1A/1B/1D}R$	≥98%

Baclofen



Baclofen is a GABA derivative that acts as an agonist at $GABA_B$ receptors; it is commonly used in animal models to examine the effects of GABAergic neurotransmission in a variety of fields, including substance dependence, spasticity, pain, and feeding behavior[1,2]. The analgesic effect of baclofen is mediated by its $GABA_B$ receptor activation, resulting in a presynaptic block of action potentials induced by mechanical or thermal stimuli that decreases frequency and amplitude of excitatory post-synaptic currents[3,4]. Baclofen is used clinically to promote abstinence in alcohol dependent subjects, mediating alcohol craving during withdrawal[5]. Baclofen is also delivered intrathecally to treat spasticity and dystonia[6].

1. Miner P, et al. Brain Res. 2010 Oct 8; 1355:86-96.
2. Kumru H, et al. Eur J Pain. 2013 Aug;17(7):1039-47.
3. Fukuhara K, et al. Eur J Neurosci. 2013 Aug 20. [Epub ahead of print].
4. Levy RA, et al. J Pharmacol Exp Ther. 1977 Aug;202(2):437-45.
5. Brennan JL, et al. Clin Pharmacol. 2013 Jul 3;5:99-107.
6. Uchiyama T, et al. Neurol Med Chir (Tokyo). 2012;52(7):463-9.

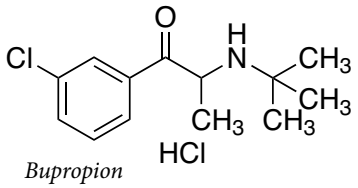


Neurotransmitter Modulators

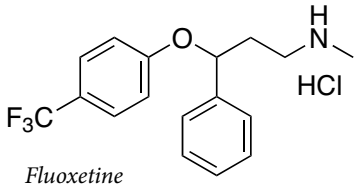
Bupropion

Bupropion is an antidepressant that is also commonly used as a smoking cessation aid. This compound has additional therapeutic potential, as it promotes modest weight loss, shows efficacy as an ADHD treatment, and may also treat neuropathic pain[1,2,3]. Bupropion is a DA and NE reuptake inhibitor, decreasing the firing rate of NE neurons due to activation of their inhibitory somato-dendritic α_2-adrenoreceptors by circulating NE; bupropion's effects on NE reuptake are stronger than its effects on DA reuptake[4]. Bupropion is also a noncompetitive antagonist at $\alpha_3\beta_2$, $\alpha_3\beta_4$, $\alpha_4\beta_2$ nAChRs, lowering channel opening probability in closed nAChRs and accelerating desensitization in open nAChRs[5,6].

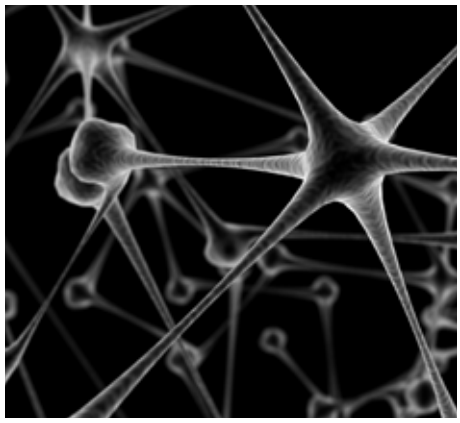
1. Li Z, et al. Ann Intern Med. 2005 Apr 5;142(7):532-46.
2. Cantwell DP. J Clin Psychiatry. 1998;59 Suppl 4:92-4.
3. Shah TH, et al. Am J Hosp Palliat Care. 2010 Aug;27(5):333-6.
4. Dong J, et al. Psychopharmacology (Berl). 2001 Apr;155(1):52-7.
5. Arias HR, et al. Int J Biochem Cell Biol. 2009 Nov;41(11):2098-108.
6. Miller DK, et al. J Pharmacol Exp Ther. 2002 Sep;302(3):1113-22.



Bupropion



Fluoxetine



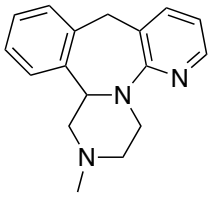
Fluoxetine

Fluoxetine is an SSRI most often used to treat mood or psychiatric disorders such as depression, OCD, bulimia nervosa, panic disorder, and PTSD. Fluoxetine's primary mechanism of action involves competitive inhibition of 5-HT reuptake by 5-HT transporters as well as inhibitory activity at 5-HT receptors and σ_1 receptors[1,2,3]. Fluoxetine can prevent cue- and stress-induced reinstatement in animal models of substance abuse. In a clinical setting, fluoxetine increases abstinence rates in former female heroin-dependent subjects when combined with naltrexone compared to naltrexone alone[4]. Additionally, this compound exhibits antiviral activity, demonstrated by its ability to reduce synthesis of coxsackievirus RNA and protein[5].

1. Apparsundaram S, et al. J Pharmacol Exp Ther. 2008 Dec;327(3):982-90.
2. Pälvimäki EP, et al. Psychopharmacology (Berl). 1996 Aug;126(3):234-40.
3. Narita N, et al. Eur J Pharmacol. 1996 Jun 20;307(1):117-9.
4. Krupitsky EM, et al. J Subst Abuse Treat. 2006 Dec;31(4):319-28.
5. Zuo J, et al. Antimicrob Agents Chemother. 2012 Sep;56(9):4838-44.

Mirtazapine

Mirtazapine is an antidepressant that also displays anxiolytic, hypnotic, orexigenic, and antiemetic properties.



Mirtazapine acts as an antagonist at 5-$HT_{2/3}$ receptors, an indirect agonist at 5-HT_1 receptors, and an antagonist at α_2 receptors, enhancing NE and 5-HT neurotransmission[1,2]. The sleep-inducing hypnotic effects are mediated through mirtazapine's inverse agonism at H_1 receptors, although tolerance to this effect develops during chronic use[3]. Clinically, mirtazapine improves the withdrawal symptom profile and aids in preventing relapse in recently abstinent substance abusers in a manner akin to other antidepressants[4,5].

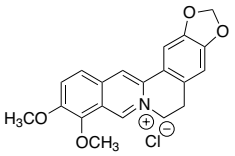
This compound also shows efficacy in altering behaviors associated with autism spectrum disorder as well as pervasive developmental disorder[6].

1. Nutt DJ. Hum Psychopharmacol. 2002 Jun;17 Suppl 1:S37-41.
2. de Boer TH, et al. Neuropharmacology. 1988 Apr;27(4):399-408.
3. Anttila SA, et al. CNS Drug Rev. 2001 Fall;7(3):249-64.
4. Liappas J, et al. J Psychopharmacol. 2004 Mar;18(1):88-93.
5. Kongsakon R, et al. Int Clin Psychopharmacol. 2005 Sep;20(5):253-6.
6. Posey DJ, et al. J Child Adolesc Psychopharmacol. 2001 Fall;11(3):267-77.

Plant- and Animal-based Products

Cat #	Product Name	Description	Purity
B1870	Berberine HCL Hydrate	Inhibits DR, OrexR, AChE, POP; activates σ_1R	≥97%
B3345	(-)-Bilobalide	Inhibits $GABA_A$R; antioxidant	≥98%
C0020	Cafestol	Activates FXR, PXR, Nrf2	≥98%
C0266	Capsaicin	Activates TRPV1	≥95%
C0278	Catechin, 99%	Inhibits MAOB, HDC; activates BDNF; antioxidant	≥99%
C8069	Curcumin	Inhibits COX; antioxidant	≥97%
D0032	Daidzein	Activates PPARα/δ/γ; antioxidant, phytoestrogen	≥97%
E6234	Epigallocatechin gallate	Inhibits CB_1R, EGFR, HER2, HAT, DNA MTase, Topo I/II, FAS	≥98%
G1853	Genipin	Induces apoptosis in glioma cells	≥98%
G3358	Ginkgolides	Activates PXR; antioxidant, protective against aβ	≥98%
G4598	Glycyrrihizin	Inhibits NAD^+, 11β-HSD; anti-inflammatory	≥93%
H8162	(-)-Huperzine A	Inhibits NMDAR, AChE	≥97%
H9861	Hypericin	Inhibits $GABA_B$R, MAO, DBH; activates AMPAR	≥97%
I7357	Isorhamnetin	Induces expression of BDNF, GDNF, NGF	≥98%
K0282	Kavalactones Mixture	Activates CB_1R, Nrf2; mod. GABA	≥98%
K0088	Kawain	Inhibits MAOB, voltage-gated L-type Ca^{2+}, Na^+ channels; activates NMDAR	≥98%
L3550	Limonin	Antioxidant, anti-inflammatory, antinociceptive	≥98%
M9368	Myristicin	Inhibits $GABA_A$R, MAO; activates $5\text{-}HT_{2A}$R; precursor to MMDA	≥97%
O0977	Octopamine HCL	Analog of NE; activates OA1/2R, CREB; modulates synaptic plasticity, feeding, Ca^{2+}, AdCyc	≥98%
P3465	Piperine	Activates TRPV1; antioxidant	≥95%
Q8016	Quercetin Dihydrate	Inhibits MAOA, RT; modulates apoptosis in glioma cells; antioxidant, anti-inflammatory	≥95%
R1776	Resveratrol	Inhibits MAOA, HSV; activates SIRT1, AMPK; antioxidant, anti-inflammatory	≥98%
R3197	Rhyncholphylline	Inhibits NMDAR, voltage-gated L-type Ca^{2+}, hERG K^+ channels	≥98%
R5874	Rosmarinic acid	Inhibits AChE, GABA-T, COX; antioxidant	≥98%
S1853	Senegenin	Increases NMDAR NR2B expression	≥98%
S9753	Synephrine	Activates 5-HTR, $\alpha_{1/2}$R, TAAR1	≥98%
T2816	L-theanine	Activates AMPAR, NMDAR; increases 5-HT, DA, GABA	≥98%

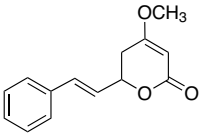
Berberine



Berberine is an alkaloid found in a variety of plants, including barberry, goldenseal, Oregon grape, Amur cork tree, and California poppy. Berberine fluoresces under ultraviolet light and is used to stain heparin in mast cells. Like many other natural products, berberine displays many beneficial effects, including immunomodulatory, anticancer, antiviral, anti-inflammatory, and antidepressant activities. Berberine is a competitive inhibitor of both AChE and prolyl oligopeptidase, enzymes important in neuropsychiatric disorders such as Alzheimer's disease, depression, schizophrenia, and anxiety[1,2,3]. In animal models of depression, berberine increases levels of 5-HT, DA, and NE and is also thought to act on σ receptors[4].

1. Huang L, et al. Bioorg Med Chem. 2010 Feb;18(3):1244-51.
2. Tarrago T, et al. ChemMedChem. 2007 Mar;2(3):354-9.
3. Kulkarni SK, et al. Phytother Res. 2010 Mar;24(3):317-24.
4. Kulkarni SK, et al. Eur J Pharmacol. 2008 Jul 28;589(1-3):163-72.

Kavalactones/Kawain

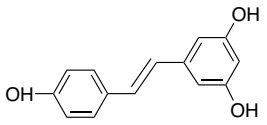


Kawain is a kavalactone found in the roots of the kava plant. Kavalactones exert a wide variety of activities which include antinociceptive, anxiolytic, hypnotic, anticonvulsant, and anti-inflammatory effects. Kavalactones shorten sleep latency and decrease awake time in sleep-disturbed rats and effectively treat short-term anxiety in humans[1,2]. In animals, these compounds also activate Nrf2, a transcription factor protective against Aβ-induced neurotoxicity in Alzheimer's disease and inhibit MPTP-induced loss of DA, tyrosine hydroxylase, and nigral neurons in models of Parkinson's disease[3,4]. Additionally, kavalactones modulate Na^+, K^+, and Ca^{2+} ion channel signaling as well as chemical and thermal pain nociception[5,6].

1. Tsutsui R, et al. J Pharmacol Sci. 2009 Nov;111(3):293-8.
2. Pittler MH, et al. Cochrane Database Syst Rev. 2003;(1):CD003383.
3. Wruck CJ, et al. Mol Pharmacol. 2008 Jun;73(6):1785-95.
4. Schmidt N, et al. Synapse. 2001 Apr;40(1):47-54.
5. Grunze H, et al. Prog Neuropsychopharmacol Biol Psychiatry. 2001 Nov;25(8):1555-70.
6. Kormann EC, et al. Pharmacol Rep. 2012;64(6):1419-26.

Resveratrol

Resveratrol is a phenolic phytoalexin found in several plants such as Japanese knotweed, soybeans, and grapes. Resveratrol is most well known for its antioxidative effects and activation of SIRT1, both contributing to its cardioprotective, anticancer, anti-aging, anti-inflammatory, and antiviral activities[1]. Resveratrol reversibly inhibits MAO as well as synaptosomal 5-HT and NE uptake, indicating potential antidepressant activity[2]. This compound displays neuroprotective activity in models of Alzheimer's disease, degrading aβ plaques, increasing brain cysteine, and decreasing brain glutathione; these effects may depend on resveratrol's activation of AMPK or proteasomes[3,4,5].



1. Mohar DS, et al. J Clin Exp Cardiolog. 2012 Nov;3(11). pii: 216.
2. Yáñez M, et al. Biochem Biophys Res Commun. 2006 Jun 2;344(2):688-95.
3. Marambaud P, et al. J Biol Chem. 2005 Nov 11;280(45):37377-82.
4. Karuppagrounder SS, et al. Neurochem Int. 2009 Feb;54(2):111-8.
5. Vingtdeux T, et al. J Biol Chem. 2010 Mar 19;285(12):9100-13.

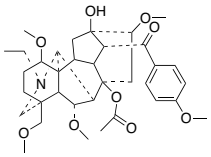
Ion Channel Modulators

Cat #	Product Name	Description	Purity
B8144	Bulleyaconitine A	Inhibits voltage-gated Na^+ channels	≥96%
B8248	Bumetanide	Inhibits NKCC1 co-transporter	≥98%
B8261	Bupivacaine	Inhibits TREK-1, voltage-gated Na^+, K^+ channels	≥98%
C0270	Carbamazepine	Inhibits voltage-gated Na^+ channels; pot. GABA	≥98%
C1644	Celecoxib	Activates voltage-dependent KCNQ (K_v7) K^+ channels; inhibits COX-2	≥98%
C3251	Cinnarizine	Inhibits T-type voltage-gated Ca^{2+} channels, D_2R, H_1R	≥98%
D3209	Diclofenac, Na^+ Salt	Activates KCNQ2/3/4 ($K_v7.2/3/4$) K^+ channels; inhibits COX, voltage-gated Na^+, KCNQ5 ($K_v7.5$) K^+ channels	≥98%
F4583	Flupirtine Maleate	Activates voltage-gated KCNQ (K_v7) channels, inhibits NMDAR	≥98%
G0106	Gabapentin	GABA analog; inhibits voltage-gated N-type Ca^{2+} channels; activates Ad1R	≥98%
I5315	Indomethacin	Inhibits COX, Ca^{2+} current; activates PPARγ	≥98%
L0349	Lamotrigine	Inhibits voltage-gated Na^+, N/P/Q/R-type Ca^{2+} channels	≥98%
L0060	Lappaconitine	Inhibits voltage-gated Na^+ channels	≥98%
L1784	Levetiracetam	Inhibits SV2A, presynaptic Ca^{2+} release	≥98%
N3322	Niflumic Acid	Inhibits voltage-gated T-type Ca^{2+}, Cl- channels, NMDAR; mod. GABAR	≥98%
O9210	Oxcarbazepine	Inhibits nAChRs, voltage-dependent Na^+, K^+ channels	≥98%
P7059	Proxymetacaine HCL	Inhibits voltage-gated Na^+ channels	≥98%
R1977	Retigabine	Activates voltage-dependent KCNQ (K_v7) K^+ channels	≥98%
V0147	Valproic Acid, Na^+	Inhibits voltage-gated Na^+, T-type Ca^{2+} channels, GABA-T, HDAC	≥98%

Bulleyaconitine

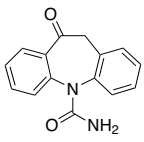

Aconitum bulleyanum

Bulleyaconitine A (BLA) is a natural product found in the *Aconitum bulleyanum* plant that exhibits analgesic and anesthetic activities. BLA has been used for several decades in China as a treatment for a variety of pain-related and inflammatory disorders. BLA inhibits voltage-dependent Na^+ channels in a use-dependent manner, reducing peak Na^+ currents during repeated stimulation in vitro and in vivo[1]. In animal models, combination of BLA with lidocaine or epinephrine reduced drug absorption and prolonged the anesthetic effect with minimal adverse effects[2]. Like other aconitines, BLA is thought to act at neurotoxin receptor site 2[3].



1. Wang CF, et al. Anesth Analg. 2008 Oct;107(4):1397-405.
2. Wang CF, et al. Anesthesiology. 2007 Jul;107(1):82-90.
3. Catterall WA, et al. Toxicon. 1985;23(3):497-504.

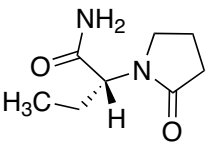
Oxcarbazapine



Oxcarbazepine (OX) is an anticonvulsant used to treat epilepsy, but also exhibits activity as a treatment for mood disorders and neuropathic pain as well[1]. Administration of OX leads to a reversible reduction in current amplitude from voltage-dependent Na^+ channels and may suppress current amplitude of delayed rectifying K^+ channels; this reduces the amplitude of action potentials and prolongs their duration[2]. This compound also inhibits Na^+ channel-dependent Glu release and produces a moderate open channel block on $\alpha_4\beta_2$ nAChRs, preventing deactivation[3,4]. Interestingly, OX may have potential as a treatment for substance abuse disorders, as it is an effective relapse prevention treatment in a clinical trial of recently abstinent alcohol-dependent subjects[5].

1. Johannessen Landmark C. CNS Drugs. 2008;22(1):27-47.
2. Huang CW, et al. Int J Neuropsychopharmacol. 2008 Aug;11(5):597-610.
3. Sitges M, et al. Neuropharmacology. 2007 Feb;52(2):598-605.
4. Di Resta C, et al. Eur J Pharmacol. 2010 Sep 15;643(1):13-20.
5. Nishimura M, et al. Am J Kidney Dis. 1992 Feb;19(2):149-55.

Levetiracetam

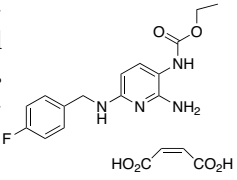


Levetiracetam is an anticonvulsant that is used to treat a wide variety of seizure disorders, including partial, myoclonic, and tonic-clonic seizures as well as mood and psychiatric disorders such as anxiety, autism, and Tourette's syndrome[1]. Levetiracetam binds to synaptic vesicle glycoprotein SV2A, inhibiting presynaptic Ca^{2+} release, reducing excitatory postsynaptic potentials, and therefore inhibiting synaptic transmission[2,3]. This compound is also under examination as a treatment for Alzheimer's disease, as it reduces memory and learning deficits, synaptic dysfunction, and hippocampal remodeling in a trangenic model[4].

1. Farooq MU, et al. Am J Health Syst Pharm. 2009 Mar 15;66(6):541-61.
2. Lynch BA, et al. Proc Natl Acad Sci U S A. 2004 Jun 29;101(26):9861-6.
3. Vogl C, et al. Mol Pharmacol. 2012 Aug;82(2):199-208.
4. Sanchez PE, et al. Proc Natl Acad Sci U S A. 2012 Oct 16;109(42):E2895-903.

Flupirtine Maleate

Flupirtine maleate is an agonist at voltage-dependent KCNQ/K_v7 K^+ channels; opening of these channels on neurons facilitates M-current generation and decreases axonal excitability[1,2]. In addition to its modulation of K^+ channels, flupirtine maleate also inhibits NMDA receptors and shifts gating of $GABA_A$-Rs to decrease circulating GABA concentrations[3,4]. Flupirtine maleate is an effective non-sedative analgesic, showing activity in neurosurgical patients[5]. In animal models, this compound also attenuates development of and reverses established pulmonary arterial hypertension, suggesting vasodilatory activity[6].



1. Fleckenstein J. J Transl Med. 2013 Feb 8;11:34.
2. Devulder J. CNS Drugs. 2010 Oct;24(10):867-81.
3. Harish S, et al. J Anaesthesiol Clin Pharmacol. 2012 Apr;28(2):172-7.
4. Klinger F, et al. Br J Pharmacol. 2012 Jul;166(5):1631-42.
5. Yadav G, et al. J Neurosurg Anesthesiol. 2013 Jun 11.
6. Morecroft I, et al. Br J Pharmacol. 2009 Aug;157(7):1241-9.

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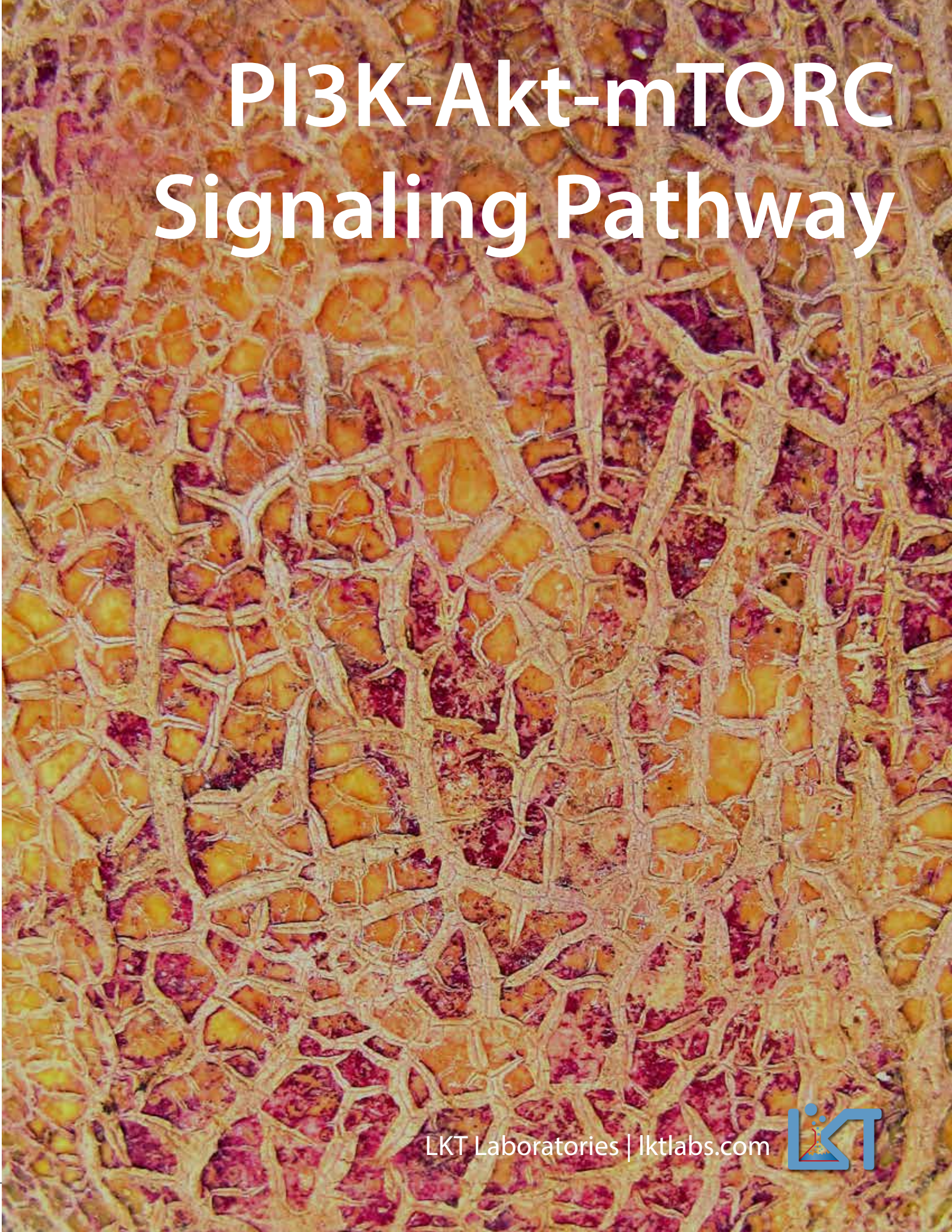
PI3K-Akt-mTORC Signaling Pathway



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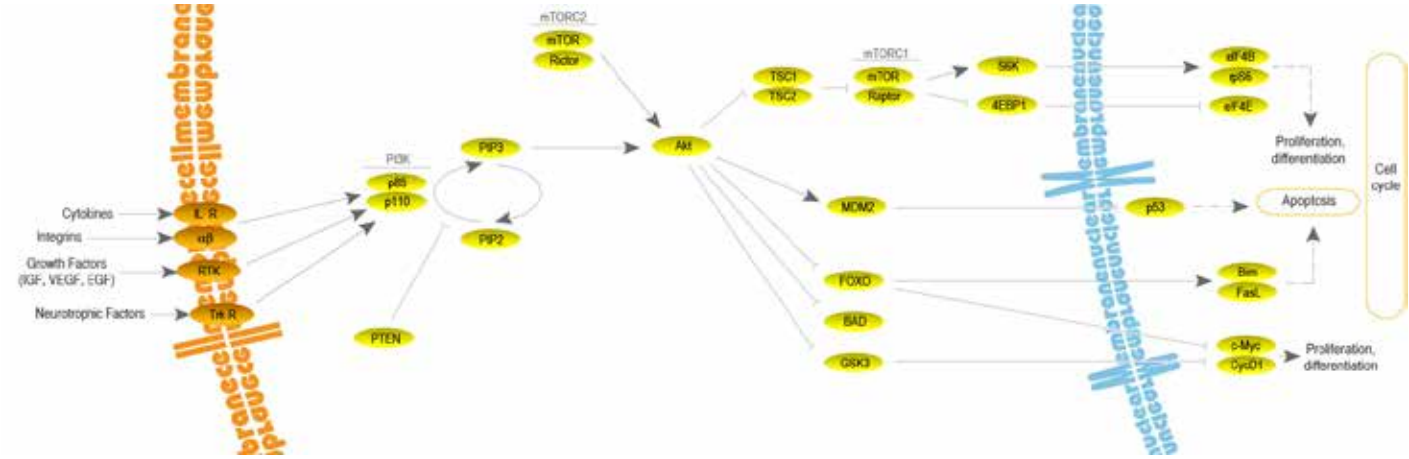
Introduction to the PI3K-Akt-mTORC signaling pathway



The PI3K-Akt-mTORC signaling pathway plays a significant role in cell survival. Activation of this pathway inhibits apoptosis, allowing for continual cell proliferation. This pathway transduces extracellular signaling from ligands of receptor tyrosine kinases in the cell membrane to nuclear transcription factors that regulate gene product synthesis. Overactive signaling by this pathway occurs in many cancers, resulting in unchecked cell growth. As a result, its proteins and receptors make excellent targets for anticancer chemotherapeutic compounds.

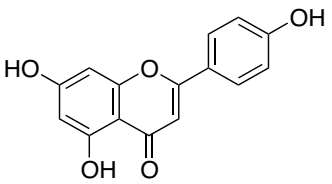
mTOR

Cat #	Product Name	Description	Purity
E4617	Aloe-emodin	Inhibits mTORC2	≥98%
A9710	AZD2014	Inhibits mTORC1/2	≥98%
A9914	AZD-8055	Inhibits mTORC1/2	≥98%
B1996	BEZ235	Inhibits mTORC1/2 and PI3K	≥98%
C8069	Curcumin	Inhibits mTORC2's phosphorylation activity	≥98%
E5057	Emodin	Inhibits mTORC2	≥95%
E8419	Everolimus	Inhibits mTORC1	≥98%
G1209	GDC-0980	Inhibits mTORC1/2 and PI3K	≥98%
G7301	GSK1059615	Inhibits mTORC1/2 and PI3K	≥98%
G7342	GSK2126458	Inhibits mTORC1/2 and PI3K	≥99%
I5440	INK128	Inhibits mTORC1/2	≥99%
M3196	MHY-1485	Activates mTORC1/2	≥98%
N8604	NVP-BGT226	Inhibits mTORC1/2 and PI3K	≥98%
O7332	OSI-027	Inhibits mTORC1/2	≥98%
P0246	Palomid 529	Inhibits mTORC1/2	≥98%
P2002	PF-04691502	Inhibits mTORC1/2 and PI3K	≥98%
P4132	PKI-402	Inhibits mTORC1/2 and p110α PI3K	≥98%
P6004	PP-242	Inhibits mTORC1/2	≥98%
R0161	Rapamycin	Inhibits mTORC1/2	≥98%
S8253	Sunitinib Malate	Inhibits mTORC1	≥98%
T6833	Triacetyl Aloe-emodin	Inhibits mTORC2	≥98%

Mammalian target of rapamycin (mTOR) is a serine/threonine protein kinase that is the catalytic subunit of complexes mTORC1 and mTORC2. mTORC1 includes mTOR, Raptor, MLST8, PRAS40, and DEPTOR and is involved in energy homeostasis signaling. Akt phosphorylates regulatory protein TSC2, inactivating the TSC1/TSC2 heterodimer and limiting its ability to inhibit mTORC1; this results in continual activation of mTORC1. mTORC2 is composed of mTOR, Rictor, MLST8, and mSin1 and plays a role in cell survival. mTORC2 is responsible for phosphorylating Akt, leading to its full activation and downstream anti-apoptotic effects. Inhibitors of mTOR and mTORC1/2 exhibit chemotherapeutic benefit in the treatment of many cancers; compounds that inhibit this kinase and its complex include sunitinib maleate, everolimus, INK128, and curcumin.

PI3K

Phosphoinositide 3-kinases (PI3Ks) compose a category of enzymes that are responsible for the formation of phosphatidylinositol-3-phosphate (PIP) products such as PIP, PIP2, and PIP3. PI3Ks are subdivided into three classes; class I PI3Ks are responsible for cell survival signaling. Class I PI3Ks are primarily composed of a p85 regulatory subunit and a p110 catalytic subunit. Activation of PI3Ks by growth factor and survival factor receptor tyrosine kinases induces production of PIP3, which together with mTOR, activates Akt. Specific isoforms of the p110 subunit of class I PI3Ks are being studied as targets in the development of new anticancer compounds, such as BYL719, GSK2636771, and CAL101.



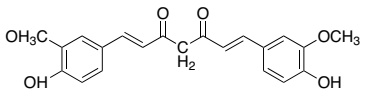
A6234 Apigenin

ID	Name	Description	Purity
A0002	A66	Inhibits p110α	≥98%
A6234	Apigenin	Inhibits PI3K (non-selective) and Akt	≥98%
B0396	BAY80-6946	Inhibits p110α	≥98%
B1996	BEZ235	Inhibits PI3K (non-selective) and mTORC1/2	≥98%
B4248	BKM120	Inhibits PI3K (non-selective)	≥98%
B9700	BYL719	Inhibits p110α PI3K	>99%, >99%ee
C0044	CAL101	Inhibits p110δ PI3K	≥98%
G1309	GDC-0941	Inhibits PI3K (non-selective)	≥98%
G1209	GDC-0980	Inhibits PI3K (non-selective) and mTORC1/2	≥98%
G7342	GSK2126458	Inhibits p110α PI3K and mTORC1/2	>99%
G7342	GSK2126458	Inhibits PI3K (non-selective) and mTORC1/2	>99%
G7346	GSK2636771	Inhibits p110β PI3K	>98.5%
G7346	GSK2636771	Inhibits p110β	>98.5%
L4796	LY294002	Inhibits PI3K (non-selective)	≥99 %
P2002	PF-04691502	Inhibits PI3K (non-selective) and mTORC1/2	≥98%
P3209	Piceatannol	Inhibits PI3K (non-selective)	>98%
P4132	PKI-402	Inhibits p110α and mTORC1/2	≥98%
P6002	PP-121	Inhibits p110α, mTORC1/2, Abl, Hck, Src, VEGFR2, PDGFR	≥98%
P9200	PX-866	Inhibits PI3K (non-selective)	≥98%
W5726	Wogonin	Inhibits PI3K (non-selective) and Akt	≥98%
X4402	XL147	Inhibits PI3K (non-selective)	≥98%
Z7477	ZSTK474	Inhibits PI3K (non-selective)	≥98%

AKT

ID	Name	Description	Purity
A9601	AZD5363	Inhibits Akt	>99%, >99%ee
C0252	Canertinib 2HCl	Indirectly inhibits Akt (via VEGFR)	≥98%
C8069	Curcumin	Inhibits phosphorylation of Akt and indirectly inhibits Akt (via Notch-1)	≥97 %
D0006	Dacomitinib	Indirectly inhibits Akt (via EGFR)	>99%
G0104	Gabexate Mesylate	Indirectly inhibits Akt (via PTEN)	≥98%
G0248	Gambogic Acid	Indirectly inhibits Akt (via EGFR)	≥98%
G1200	GDC-0068	Inhibits Akt	≥99%
G5772	Goserelin	Indirectly inhibits Akt	≥98%
G7242	GSK-690693	Inhibits Akt	≥98%
M4000	MK2206	Inhibits Akt	≥99%
N3577	Nitidine Chloride	Indirectly inhibits Akt	≥98%
P1845	Pelitinib	Indirectly inhibits Akt (via EGFR)	≥98%
P1969	Perifosine	Inhibits Akt	≥98%
P3076	PHT-427	Inhibits Akt and PDK1	≥98%
P3209	Piceatannol	Indirectly inhibits Akt (via PI3K, insulin receptor)	>98%
S8098	SU-1498	Indirectly inhibits Akt (via VEGFR2)	>98%
T0152	Tandutinib	Indirectly inhibits Akt (via c-Kit)	≥98%
T2936	Thioridazine HCl	Indirectly inhibits Akt (via FAK, αvβ3 integrin)	≥98%
W3576	Withaferin A	Indirectly inhibits Akt (via Notch-1, HSP90)	≥98%

Akt, also known as protein kinase B, is a serine/threonine protein kinase involved in anti-apoptotic signaling. Akt phosphorylates and inactivates pro-apoptotic protein Bad and alters IkB kinase activity to allow NF-kB activation and subsequent expression of anti-apoptotic gene products. Akt activation can be prevented by tumor suppressor PTEN's dephosphorylation of PIP3. Activity of Akt is associated with cancer progression and tumor development. Inhibitors of Akt include MK2206, GDC-0068, and AZD5363.



C8069 Curcumin

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- Tyrosine kinase inhibitors
- Serine/threonine kinase inhibitors
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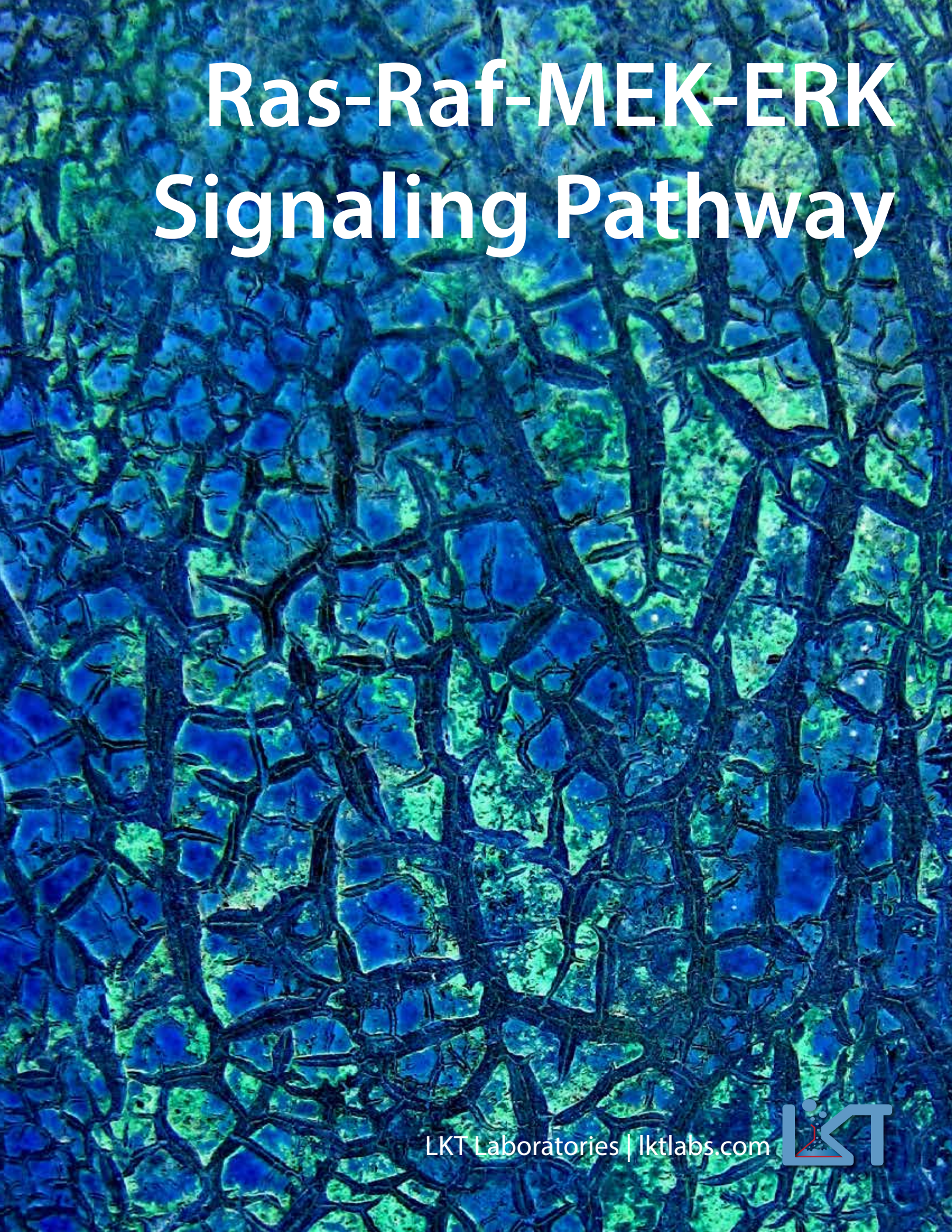
Ras-Raf-MEK-ERK
Signaling Pathway
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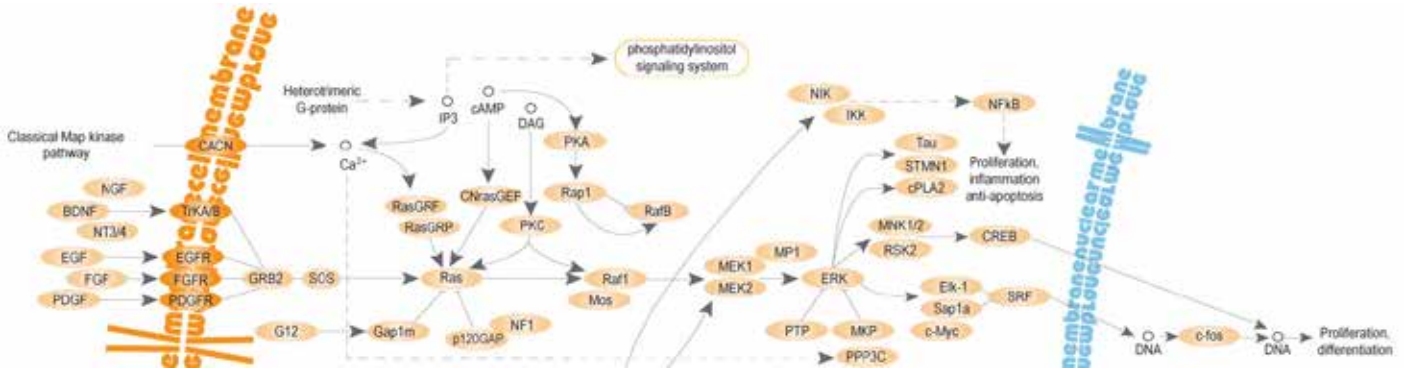
Introduction to the Ras-Raf-MEK-ERK signaling pathway



The Ras-Raf-MEK-MAPK-ERK signaling pathway plays a significant role in cell differentiation, proliferation, and survival. This pathway transduces extracellular signaling from ligands of receptor tyrosine kinases in the cell membrane to nuclear transcription factors that regulate gene product synthesis. Mutations in proteins of this pathway are found in many cancers, resulting in overactive signaling and unchecked cell growth. As a result, its proteins and receptors make excellent targets for anticancer chemotherapeutic compounds.

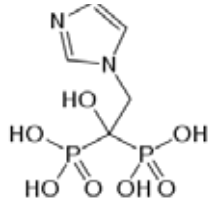
MEK

ID	Name	Description	Purity
A6971	ARRY-162	Inhibits MEK1/2	≥99%
A7203	AS-703026	Inhibits MEK1/2	≥98%
A9715	AZD8330	Inhibits MEK1/2	≥98%
G1210	GDC-0623	Inhibits MEK1/2	≥98%
G7440	GSK1120212	Inhibits MEK1/2	≥98%
P1200	PD184352	Inhibits MEK1/2	≥98%
P1202	PD325901	Inhibits MEK1/2	≥98%
R1217	RDEA119	Inhibits MEK1/2	≥98%
S1846	Selumetinib	Inhibits MEK1/2	≥98%

MEK, or mitogen-activated protein kinase kinase (MAP2K), is a family of tyrosine/threonine kinases activated by Raf responsible for phosphorylating ERK and other MAPK proteins. MEK enzymes have two primary isoforms, MEK1 and MEK2. These kinases may also be mutated in some forms of cancer, allowing continual and unregulated activation. Compounds that target MEK1/2 include RDEA119, ARRY-1662, selumetinib, and PD-325901.

Ras

ID	Name	Description	Purity
H9716	(E,Z)-4-Hydroxytamoxifen	Increases degradation of K-Ras	>97%
K5604	Kobe 0065	Inhibits Ras	≥98%
K5605	Kobe 2602	Inhibits Ras	≥98%
N5605	Nobiletin	Indirectly inhibits Ras (via PKC)	≥97%
Z5744	Zoledronic Acid	Inhibits prenylation of Ras	≥98%



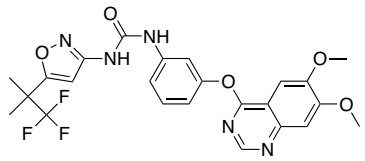
Z5744 Zoledronic Acid

Ras family proteins are small GTPases, much like Rho, Ran, and Arf, which control other cellular processes like nuclear and vesicular transport signaling. Ras proteins are typically activated by receptor tyrosine kinases such as growth factor receptors, and ephrin receptors. K-Ras, H-Ras, and N-Ras are specific proteins within the Ras family that propagate growth factor signaling through activation of downstream targets such as Raf and PI3K. Overactive mutant isoforms of K-Ras have been implicated in the development of colorectal and pancreatic cancers. Activating mutations in H-Ras have been associated with the progression of bladder cancer. Similarly, N-Ras mutations are often found in melanomas and in thyroid cancer. Ras family proteins are targeted by treatments such as zoledronic acid and nobiletin.

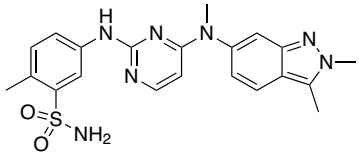
RAF

Raf proteins are serine/threonine kinases that transduce signals from Ras proteins and amplify them using the MAPK signaling cascade. The three primary Raf proteins include A-Raf, B-Raf, and C-Raf (Raf-1). Mutations occasionally occur in A-Raf and C-Raf kinases, but mutant forms of B-Raf play a significant role in the development of many cancers. In B-Raf, V599 and V600 compose part of the activation loop. Typically these residues are responsible for keeping B-Raf in an inactive conformation until the enzyme is phosphorylated, but mutations such as V599K or V600E destabilize these interactions, inducing activation and resulting in potentially uncontrolled downstream signaling and cell growth. In recent research, Raf proteins are under exploration as targets for new chemotherapeutics such as dabrafenib and vemurafenib.

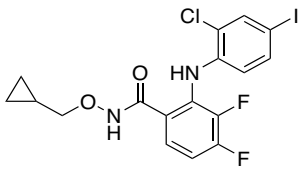
ID	Name	Description	Purity
A9662	AZ628	Inhibits V600E B-Raf, C-Raf (Raf-1)	≥96%
C1660	CEP-32496	Inhibits V600E B-Raf	≥98%
D0004	Dabrafenib	Inhibits WT B-Raf, V600E/K/D B-Raf, C-Raf (Raf-1)	≥98%
D0005	Dabrafenib Mesylate	Inhibits WT B-Raf, V600E/K/D B-Raf, C-Raf (Raf-1)	≥98%
G1208	GDC-0879	Inhibits V600E B-Raf	≥96%
G8850	GW5074	Inhibits C-Raf (Raf-1)	≥98%
M4452	MLN2480	Inhibits B-Raf	≥98%
P0397	Pazopanib	Inhibits WT B-Raf	≥97%
P1200	PD184352	Inhibits V600E B-Raf	≥98%
P1202	PD325901	Inhibits V600E B-Raf	≥98%
P4492	PLX4720	Inhibits V600E B-Raf	≥98%
R0020	RAF265	Inhibits WT B-Raf, V600E B-Raf, C-Raf (Raf-1)	≥98%
R1626	Regorafenib Monohydrate	Inhibits B-Raf	>99%
S0459	SB-590885	Inhibits B-Raf	≥98%
S5868	Sorafenib	Inhibits WT B-Raf, mutant B-Raf, C-Raf (Raf-1)	≥98%
S5869	Sorafenib Tosylate	Inhibits WT B-Raf, mutant B-Raf, C-Raf (Raf-1)	≥99 %
T0140	TAK-632	Inhibits all Raf isoforms	≥98%
V1668	Vemurafenib (PLX4032)	Inhibits V600E B-Raf	≥98%



C1660 CEP-32496



P0397 Pazopanib



P1200 PD184352

ERK

ID	Name	Description	Purity
A6234	Apigenin	Indirectly inhibits ERK1/2	≥98%
B3573	Bisdemethoxycurcumin	Indirectly inhibits ERK1/2	≥98 %
C0252	Canertinib 2HCl	Indirectly inhibits ERK1/2 (via VEGFR)	≥98%
C8500	CV-65	Indirectly inhibits ERK5, JNK, p38 MAPK	≥60%
C8501	CV-66	Indirectly inhibits ERK5, JNK, p38 MAPK	≥95%
C8502	CV-70	Indirectly inhibits ERK5, JNK, p38 MAPK	≥90%
D1850	Demethoxycurcumin	Indirectly inhibits ERK1/2	≥98%
N3577	Nitidine Chloride	Indirectly inhibits ERK1/2	≥98%
N5605	Nobiletin	Indirectly inhibits ERK1/2 (via PKC, Ras)	≥97%
O4556	Olomoucine	Indirectly inhibits ERK1/2	≥98%
P1845	Pelitinib	Indirectly inhibits ERK1/2 (via EGFR)	≥98%
S8098	SU-1498	Inhibits pERK1/2 (directly and via VEGFR2)	≥98%
T0253	Tangeretin	Indirectly inhibits ERK1/2	≥98%
V9201	VX-11e	Inhibits ERK2	≥98%

ERKs, or extracellular signal-related kinases, are classical MAP kinases that receive signals from MEK and other proteins involved in the MAPK cascade, causing dowstream activation of transcription factors such as c-Fos, c-Myc, and ELK1. These transcription factors regulate the production of gene products involved in meiosis, mitosis, and cell differentiation. Signaling involving ERK1 or ERK2 plays a major role not just in the development and progression of cancers, but also in migraines and mood disorders such as schizophrenia and bipolar disorder. Inhibitors of ERK enzymes include CV65, VX-11e, SU-1498, nobiletin, and olomoucine.

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- Natural products
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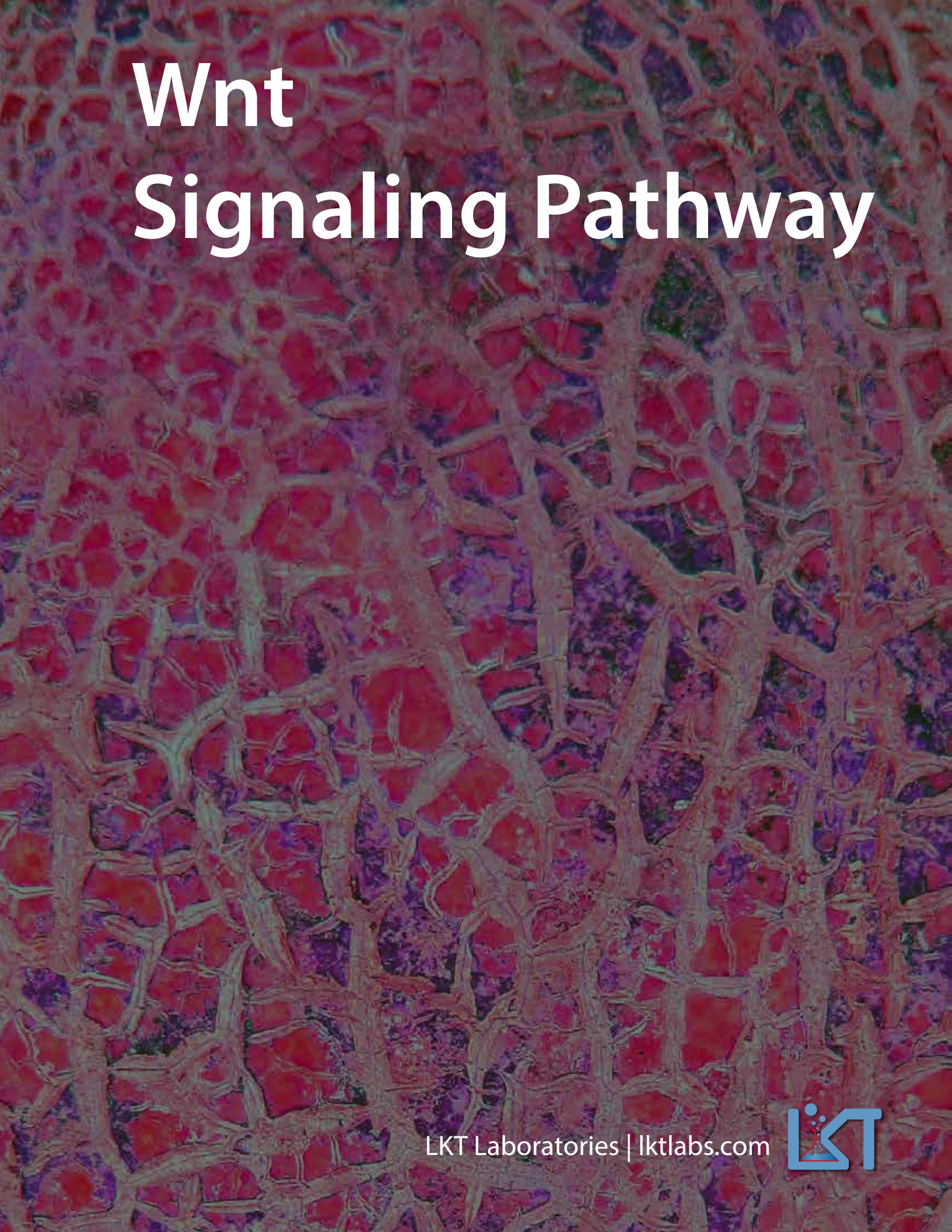
Wnt Signaling Pathway



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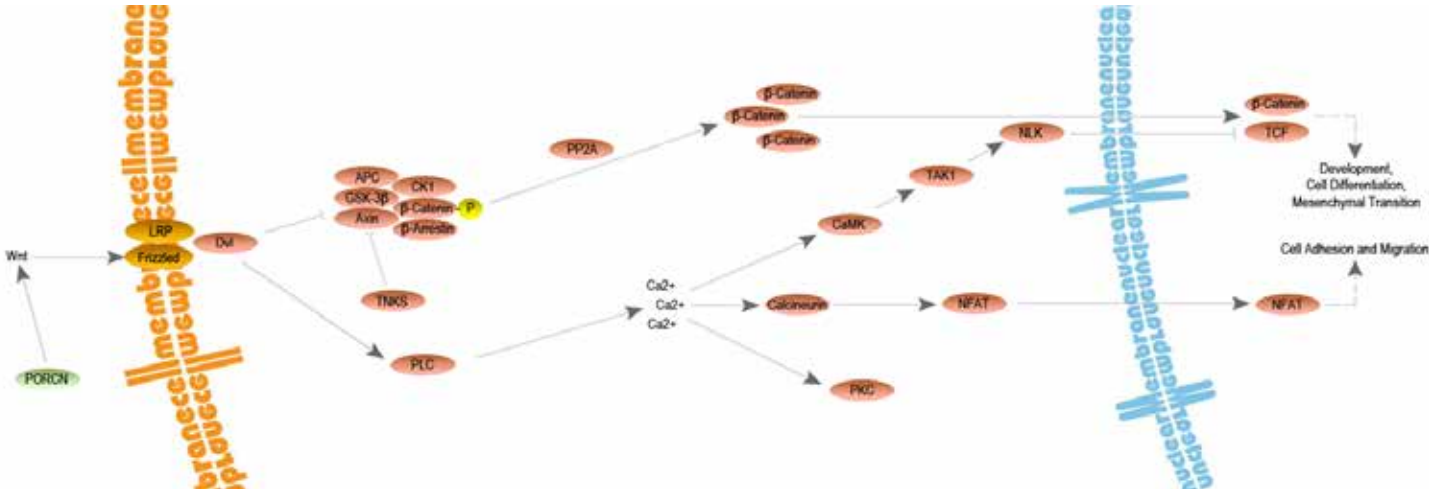
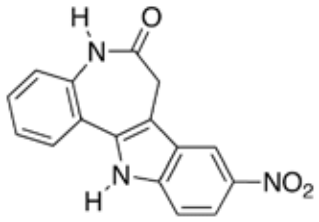
Introduction to the Wnt signaling pathway



The family of Wnt signaling pathways includes three pathways, all stimulated through the binding of Wnt to the surface receptor Frizzled. The canonical Wnt signaling pathway transduces the signal from Frizzled to Dishevelled, a protein that further signals to a destruction complex bound to β-catenin. Unless stimulated, this complex of proteins induces proteasomal degradation of β-catenin. When activated, β-catenin is dephosphorylated and enters the nucleus to stimulate cell proliferation, cell differentiation, and development. The non-canonical/calcium-dependent Wnt signaling pathway is involved in regulation of intracellular calcium levels and cell adhesion. Here, Dishevelled interacts with a trimeric G-protein to stimulate downstream release ofcalcium, which activates calcineurin and CaMK, leading to activation of the transcription factor NFAT. Lastly, the non-canonical/planar cell polarity pathway (not shown) transduces signals from Dishevelled to Rho and ROCK to stimulate actin polymerization and cytoskeleton restructuring. Several components in these pathways play a role in the development of several diseases, including cancer and type 2 diabetes.

Wnt Signaling Modulators

ID	Name	Description	Purity
A4577	Alsterpaullone	Indirectly activates Wnt signaling	≥98%
B3573	Bisdemethoxycurcumin	Indirectly inhibits Wnt signaling	≥98%
C2945	Chlorophyllin sodium copper salt	Indirectly inhibits Wnt signaling	≥99%
K9600	KY-02111	Indirectly inhibits Wnt signaling	≥98%
M9367	Myricetin	Indirectly activates Wnt signaling	≥98%
N1982	Neuromedin U, rat	Indirectly activates Wnt signaling	≥95%
O1078	Octreotide acetate	Indirectly inhibits Wnt signaling	≥98%



A4577 Alsterpaullone

Tankyrase Inhibitors

ID	Name	Description	Purity
J8800	JW55	Directly inhibits tankyrase	≥98%
X0384	XAV-939	Directly inhibits tankyrase	≥95%

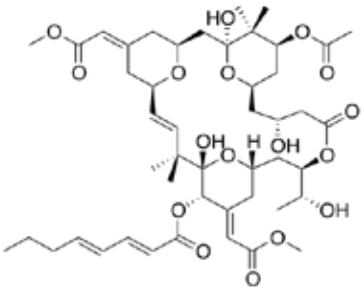
Tankyrases are members of the Poly (ADP-ribose) polymerase (PARP) family of proteins that contain ankyrin repeats, an oligomerization domain, and a PARP catalytic domain. Tankyrases interact with Axin, one component of the β-catenin destruction complex, inducing Axin degradation degradation through the ubiquitin-proteasome pathway. Inhibition of tankyrases stabilizes Axin, stimulating destruction of β-catenin and preventing downstream activation of processes such as cell differentiation and epithelial-to-mesenchymal transition.

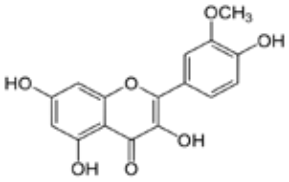
β-Catenin Inhibitors

β-Catenin is a protein that regulates cell adhesion, differentiation, and development. β-Catenin is a component of the cadherin protein complex that amplifies signal transduction and stimulates gene transcription in the Wnt signaling pathway. This protein binds transcriptions factors to stimulate development of entire body regions in early embryo stages. It also plays a role in the maintenance of stem cell pluripotency and differentiation. In later development stages, it induces epithelial-to-mesenchymal transition. Mutations in β-catenin are commonly implicated in cancers such as hepatocellular carcinoma, colorectal cancer, ovarian carcinoma, and lung cancer. Often, these cancers feature loss-of-function mutations that prevent regulation of β-catenin and allow it to stimulate gene transcription unchecked.

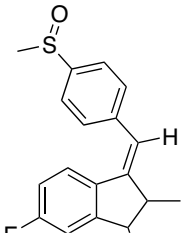
ID	Name	Description	Purity
A4931	3-Aminobenzamide	Indirectly inhibits β-catenin	≥97%
B6998	Bryostatin 1	Indirectly inhibits β-catenin	≥98%
E7309	Esculetin	Directly inhibits β-catenin	≥98%
I7357	Isorhamnetin	Indirectly inhibits β-catenin	≥98%
S8145	Sulindac	Indirectly inhibits β-catenin	≥98%
S8147	Sulindac Sulfide	Indirectly inhibits β-catenin	≥98%
S8146	Sulindac Sulfone	Indirectly inhibits β-catenin	≥97%
T1777	S,S-(+)-Tetrandrine	Indirectly inhibits β-catenin	≥98%
T7035	Triptolide	Indirectly inhibits β-catenin	≥98%
T7056	Troglitazone	Indirectly inhibits β-catenin	≥97%

B6998 Bryostatin I

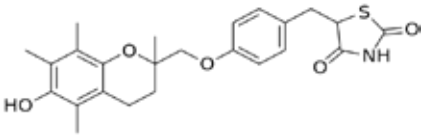




I7357 Isorhamnetin



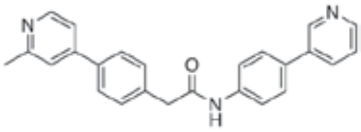
S8145 Sulindac



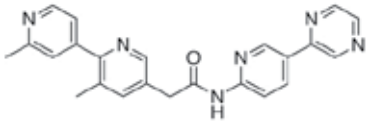
T7056 Troglitazone

PORCN Inhibitors

ID	Name	Description	Purity
C0800	C59	Directly inhibits PORCN	≥98%
I9060	IWP-2	Directly inhibits PORCN	≥98%
L2540	LGK-974	Directly inhibits PORCN	≥98%



C0800 C59



L2540 LGK-974

PORCN is a member of the membrane-bound O-acyl transferase (MBOAT) family that regulates Wnt signaling. Palmitoylation by PORCN is required for Wnt to be released from the Golgi to the cell surface and also to bind to the Frizzled receptor. Without PORCN, Wnt ligands are not secreted and embryos fail to gastrulate. PORCN is a key protein required for embryonic development, but inhibiting it can also limit Wnt-driven signaling of β-catenin and other proteins that play roles in the development of diseases such as cancer.

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Amyotrophic Lateral Sclerosis

Amyotrophic lateral sclerosis (ALS) is a neurodegnerative disease that affects motor neurons. Degeneration of motor neurons causes muscle weakness and atrophy, often leading to respiratory failure. Currently, riluzole is the only treatment that is approved to treat ALS. Several other compounds show potential benefit in cellular and animal models. These products display neuroprotective activity by improving motor neuron survival, preserving motor function, slowing disease progression, and increasing life span. Below are a variety of promising compounds carried by LKT Laboratories.

Product ID	Product Name	Description
L5751	Lomerizine Dihydrochloride	L-type and T-type Ca^{2+} and TRP antagonist.
N0160	NAP Peptide	Octapeptide derived from activity-dependent neuroprotective protein (ADNP).
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^{+} channel blocker, GLT-1 modulator.
F0275	Fasudil Hydrochloride	ROCK inhibitor.
P0013	P7C3	Aminopropyl carbazole, neuroprotective.
P0109	P7C3A20	Fluorinated aminopropyl carbazole, neuroprotective.
P0110	(R)-P7C3-OMe	Methoxy aminopropyl carbazole, neuroprotective.
K1655	Kenpaullone	GSK-3β, HGK, and CDK inhibitor.
D3329	7,8-Dihydroxyflavone Hydrate	BDNF mimetic, TrkB agonist.
T3568	Tirasemtiv	Fast skeletal muscle troponin complex activator.

Huntington's Disease

Huntington's disease is a neurodegenerative disorder that involves motor deficits and cognitive decline. The genetic basis for Huntington's disease is a string of CAG repeats found in the Huntingtin gene that results in production of a mutant gene associated with lack of motor coordination, cognitive decline, and disruptive mood disorders. Current therapies for Huntington's disease reduce symptom severity but are unable to cure the disease. Several compounds such as those listed below have shown promise in cellular and animal models of Huntington's disease as potential treatments to reduce mutant Huntingtin gene production, improve cell survival, or improve disease pathology in other ways.

Product ID	Product Name	Description
C9673	Cysteamine Hydrochloride	Aminothiol, CoA component; ulcer inducer.
C1637	Ceftriaxone Sodium	β-lactam cephalosporin; penicillin binding protein inhibitor.
V1854	Venlafaxine Hydrochloride	SERT, NET, MAO inhibitor.
M3476	Mithramycin	Polyketide; DNMT 1 inhibitor, RNA synthesis inhibitor.
A4002	AK-7	SIRT2 inhibitor.
I1418	Idebenone	Synthetic quinone CoQ analog, promotes mitochondrial respiration and ATP production.
M3353	Minocycline Hydrochloride	Tetracycline; protein translation inhibitor, MMP inhibitor.

Multiple Sclerosis

Multiple sclerosis (MS) is an autoimmune disease characterized by the loss of oligodendrocytes and demyelination of neurons. MS is associated with a wide variety of nervous system dysfunction, including pain, muscle spasms, ataxia, and visual complications. Currently, less than ten therapies have been approved for human use in the management of MS, including dimethyl fumarate, fingolimod, mitoxantrone, glatiramer acetate, and teriflunomide. Several other compounds are currently under investigation, displaying potential benefit in experimental autoimmune encephalitis (EAE), an animal model of MS. For a variety of approved and investigatory products, please consider LKT Laboratories as your new source!

Product ID	Product Name	Description
C9609	Cyclophosphamide Monohydrate	Nitrogen mustard, DNA alkylator.
R1876	all-trans-Retinol	Diterpene component of vitamin A, differentiation inducer; RAR and RXR agonist.
P6818	Prednisolone	Cortisol derivative; glucocorticoid agonist.
P7012	Prednisolone Sodium Phosphate	Water-soluble cortisol derivative; glucocorticoid agonist.
E7377	Estriol	Endogenous steroid hormone, estradiol metabolite, used in HRT; ER agonist.
C4402	Cladribine	Nucleoside (deoxyadenosine) analog; DNA chain terminator, ribonucleotide reductase and DNA polymerase inhibitor.
L8248	Lumiracoxib	NSAID; COX-2 inhibitor.
M0374	Masitinib	PDGFR and c-Kit inhibitor.
F3454	Fingolimod Hydrochloride	Sphingosine 1-phosphate antagonist.
G4781	Glucoraphanin Potassium Salt	Glucosinolate found in cruciferous vegetables.
P3592	Pixantrone Dimaleate	Aza-anthracenedione, DNA intercalator; topoisomerase II inhibitor.
E4418	β-Elemene	Sesquiterpene found in various plant sources.
R1877	all-trans-Retinol, high purity	Diterpene vitamin A; RAR/RXR agonist.
D1720	Deferiprone	Iron chelator.
D3448	Dimethyl Fumarate	Fumaric acid methyl ester; nAChR agonist, indirect Nrf2 activator.
C0169	Carbenoxolone Disodium	Synthetic derivative of glycyrrhizin; connexin and 11β-hydroxysteroid dehydrogenase inhibitor.
D3232	3,3'-Diindolylmethane	Indole found in cruciferous vegetables; AhR agonist.
G3456	Ginsenoside Rd	Triterpene saponin found in species of *Panax*; 26S proteasome inhibitor, TRPM7 antagonist.
L8377	Luteolin	Flavonoid found in various plant sources; DAT and NET potentiator, HSP90, IGF-1R, PDE inhibitor, potential α2-adrenergic antagonist.
M0278	Matrine	Alkaloid found in *Sophora*.
A7085	Arvanil	CB1 agonist, TRPV1 agonist.
K1655	Kenpaullone	GSK-3β, HGK, and CDK inhibitor.
S1970	Serum Thymic Factor	Synthetic peptide hormone.
T3093	Thymopentin	Synthetic peptide, thymopoietin analog.
T3099	Thymus Factor	Endogenous peptide involved in immune signaling.
A7578	Astragaloside IV	Found in *Astragalus membranaceus*.
T0249	Tamibarotene	RARα/β agonist.
F0275	Fasudil Hydrochloride	ROCK inhibitor.
C0167	Carbenoxolone	Synthetic glycyrrhetinic acid derivative; 11β-HSD inhibitor, gap junction connexin channel blocker.
A5303	Anatabine	Minor tobacco alkaloid found in *Solanaceae* family plants.
A7202	AS-604850	p110γ PI3K inhibitor.
A7204	AS-605240	p110δ PI3K inhibitor.
M3379	Mitoxantrone Dihydrochloride	Anthracenedione; DNA intercalator, Pim-1 inhibitor.
A9812	AZD-1480	JAK1/2 inhibitor.
M1444	MDL 29951	GPR17 agonist, NMDA and fructose 1,6-bisphosphatase inhibitor.

Alzheimer's Disease

Product ID	Product Name	Description
C8069	Curcumin	Diarylheptanoid found in *Zingiberaceae*.
F1669	Ferulic Acid	Hydroxycinnamic acid found in various plant sources, metabolite of verbascoside.
G1652	Genistein	Phytoestrogen, isoflavone found in various plant sources including soy.
I0481	Ibuprofen	NSAID; COX-1/2 inhibitor.
I5213	Indole-3-carbinol	Indole, glucosinolate found in cruciferous vegetables.
M1745	Melatonin	Endogenous hormone involved in circadian rhythms; MT agonist.
M9367	Myricetin	Flavonol found in fruits and vegetables; COMT inhibitor.
N0068	Naringenin	Flavonone found in species of *Citrus*.
R1776	Resveratrol	Phenolic phytoalexin found in soy, grapes, and peanuts; SIRT1 activator, MAO inhibitor.
R8076	Rutin Hydrate	Flavonoid glycoside found in fruit, asparagus, and buckwheat.
S8044	R,S-Sulforaphane	Synthetic ITC.
E6234	Epigallocatechin gallate	Flavanol found in *Camilla* (green tea); AhR antagonist, STAT3, α-amylase, α-glucosidase inhibitor.
S3345	Silymarin	Flavonolignan mixture found in *Silybum* (milk thistle) seeds.
B3358	Biochanin A	Phytoestrogen, flavonoid found in clover, soy, and alfalfa; potential PPARα and PPARγ agonist.
G3354	Ginkgolide A	Terpene lactone found in *Ginkgo*; GSK-3β inhibitor, potential PXR agonist.
C0265	Carnosic Acid	Diterpene found in *Rosmarinus*.
T0154	Tanshinone IIA	Found in *Salvia*; MAG lipase inhibitor.
B1870	Berberine Hydrochloride Hydrate	Isoquinoline alkaloid found in various plant sources; prolyl oligopeptidase and AChE inhibitor, potential σ modulator.
B6998	Bryostatin 1	Macrolide lactone found in *Bugula*; TLR4 activator, PKC modulator.
G3453	Ginsenoside Rh2	Triterpene saponin found in *Panax*.
P2815	Phenylbutyrate Sodium	HDAC inhibitor.
M3321	Mifepristone	Synthetic steroid, contraceptive; PR and glucocorticoid antagonist.
P8117	Puerarin, 99%	Isoflavone found in *Pueraria*; 5-HT2C and GABA-A antagonist.
P8118	Puerarin	Isoflavone daidzein derivative found in *Pueraria*; GABA-A potentiator, 5-HT2C antagonist.
T1777	S,S-(+)-Tetrandrine	Benzylisoquinoline alkaloid found in *Stephania*; voltage-gated Ca^{2+} channel blocker.
E6231	(-)-Epicatechin	Flavanol found in *Camilla* (green tea).
R5874	Rosmarinic Acid	Caffeic acid ester found in *Melissa, Salvia,* and *Rosmarinus*; GABA transaminase and Fyn kinase inhibitor.
R3220	Rifampicin	Ansamycin; bacterial DNA-dependent RNA polymerase inhibitor, RNA synthesis inhibitor.
N0069	Naringin	Flavanone glycoside found in *Citrus*; SERM.
D3330	Dihydrotanshinone	Found in *Salvia*; fatty acid synthase and AChE inhibitor, mineralocorticoid and glucocorticoid antagonist.
G1650	Geniposide	Iridoid glycoside found in *Gardenia*.
N5778	Notoginsenoside R1	Triterpene saponin found in species of *Panax*.
P7318	Pseudoginsenoside F11	Saponin found in species of *Panax*; PPARγ agonist.
I0482	S-(+)-Ibuprofen	Optically active isomer of ibuprofen, NSAID; COX-1/2 inhibitor.
I0901	Icariin	Flavonol glycoside found in *Epimedium*; PDE5 inhibitor.
M0125	Magnolol	Lignan found in *Magnolia*; GABA-A potentiator.
M1749	Memantine Hydrochloride	D2 agonist, NMDA, 5-HT3, α7 nAChR antagonist.
P0297	Paroxetine Hydrochloride Hemihydrate	FIASMA, SERT and NET inhibitor, mAChR antagonist.
A5326	Aniracetam	AMPA positive allosteric modulator, D2, 5-HT2A, nAChR agonist.
G0246	Galantamine Hydrobromide	Alkaloid found in *Galanthus, Narcissus, Leucojum*, and *Lycoris*; AChE inhibitor, α7 nAChR agonist, mAChR agonist.
I1418	Idebenone	Synthetic quinone CoQ analog, promotes mitochondrial respiration and ATP production.
N3448	Nimodipine	L-type Ca^{2+} channel blocker.
S0930	Schisantherin A	Dibenzocyclooctadiene found in *Schisandra*.
S1058	Scopolamine N-butylbromide	Tropane alkaloid found in *Solanaceae* plants; M1 mAChR antagonist.
S1059	Scopolamine Hydrobromide Trihydrate	Tropane alkaloid found in *Solanaceae* plants; M1 mAChR antagonist.
S1609	Securinine	Alkaloid found in *Securinega*; potential GABA-A antagonist.
A4577	Alsterpaullone	GSK-3 and CDK inhibitor.
A6234	Apigenin	Flavonoid found in various plant sources; GABA-A positive modulator, potential microtubule depolymerization inducer.
H8162	(-)-Huperzine A	Sesquiterpene alkaloid found in *Huperzia serrata*; AChE inhibitor, NMDA antagonist.

Product ID	Product Name	Description
K0282	Kavalactones Mixture	Mixture of kavalactones found in *Piper methysticum* (kava plant).
L1660	Leptin (22-56), human	Endogenous peptide fragment, involved in feeding behavior and energy homeostasis; leptin agonist.
M7528	α-Melanocyte Stimulating Hormone	Endogenous peptide hormone, derived from POMC, involved in energy homeostasis and melanin production; MCR agonist.
P3465	Piperine	Alkaloid found in black and long peppers; TRPV1 agonist.
D3355	Diosgenin	Steroidal saponin found in *Dioscorea*.
G3359	Ginkgolide J	Terpene lactone found in *Ginkgo*.
H2980	Humanin, human	Endogenous peptide; FPRL1/2 agonist.
P1869	Perindopril Erbumine	ACE inhibitor.
S8005	Substance P	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8006	Substance P (1-4)	Endogenous tachykinin neuropeptide; NK1 agonist
S8007	Substance P (1-7)	Endogenous tachykinin neuropeptide; NK1 agonist
S8008	Substance P (1-9)	Endogenous tachykinin neuropeptide; NK1 agonist
S8010	[Nle11]-Substance P	Endogenous tachykinin neuropeptide; NK1 agonist
S8012	[Sar9]-Substance P	Endogenous tachykinin neuropeptide; NK1 agonist
S8013	[Tyr8]-Substance P	Endogenous tachykinin neuropeptide; NK1 agonist
S8014	Substance P, free acid	Endogenous tachykinin neuropeptide; NK1 agonist
L1784	Levetiracetam	SV2A synaptic vesicle inhibitor.
Q8019	Quetiapine Fumarate	5-HT1A and σ1/2 agonist, 5-HT2A/2C/6/7, D1-4, histamine H1/2, M1 mAChR, α1A/1B/2C-adrenergic antagonist.
R3586	Rivastigmine Hydrogen Tartrate	AChE and BChE inhibitor.
A0919	Acetyl-L-Carnitine	Carnitine derivative, involved in energy homeostasis.
B5874	Bosutinib	Src and Abl inhibitor.
E5575	Entacapone	COMT inhibitor.
F3473	Fisetin	Flavonoid found in various plant sources; matrix metalloproteinase inhibitor, topoisomerase I and II inhibitor.
Z3444	Zileuton	5-Lipoxygenase inhibitor.
T0249	Tamibarotene	RARα/β agonist.
M0374	Masitinib	PDGFR and c-Kit inhibitor.
F3454	Fingolimod Hydrochloride	Sphingosine 1-phosphate antagonist.
V3345	Vildagliptin	DPP4 inhibitor.
H9814	25-Hydroxyvitamin D2	Vitamin D2, ergocalciferol metabolite; VDR agonist.
D5753	Donepezil Hydrochloride	GSK3 and AChE inhibitor, potential σ1 agonist.
M0255	Manzamine A	Alkaloid found in marine sponges; vacuolar ATPase uncoupler, GSK3 inhibitor.
G4798	Glycitein	Aglycone isoflavone found in soy and red clover.
I4961	Imperatorin	Furocoumarin; voltage-gated Na^+ channel blocker, AChE and BChE inhibitor.
T0394	(+)-Taxifolin	Catechol-type flavonoid; fatty acid synthesis inhibitor.
N5605	Nobiletin	Polymethoxylated flavone found in citrus fruits; potential AMPA positive modulator.
I7256	Isobavachalcone	Prenylated chalcone flavonoid found in *Psoraleae*; cholesterol acyltransferase inhibitor.
P3592	Pixantrone Dimaleate	Aza-anthracenedione, DNA intercalator; topoisomerase II inhibitor.
F1670	Ferulic Acid Methyl Ester	Hydroxycinnamic aicd, verbascoside metabolite, ion chelator.
P3563	Piperlonguminine	Isolated from *Piper longum*.
C8069	Curcumin	Diarylheptanoid found in *Zingiberaceae*.
D1850	Demethoxycurcumin	Curcumin derivative; AMPK activator, STAT3 and eIF4E-bp3 inhibitor, potential AChE and EGFR inhibitor.
B1992	Bexarotene	RXR agonist.
L5624	Loganin	Iridoid glucoside found in *Cornus officialis*; β-secretase inhibitor.
A0922	Acetyl-L-Carnitine Hydrochloride	Carnitine derivative, involved in energy homeostasis.
D1720	Deferiprone	Iron chelator.
G9648	12-Methyl Gymnodimine	Cyclic imine marine toxin found in *Karenia*; nAChR antagonist.
S6236	13-Desmethyl Spirolide C	Cyclic imine marine toxin found in *Alexandrium*; nAChR and mAChR antagonist.
A5303	Anatabine	Minor tobacco alkaloid found in *Solanaceae* family plants.
A4000	AK-1	SIRT2 inhibitor.
D3349	Dimebon Dihydrochloride	AMPK activator, L-type Ca^{2+} channel and NMDA, histamine H1/2, α-adrenergic, 5-HT2C/5A/6 receptor antagonist.
D3329	7,8-Dihydroxyflavone Hydrate	TrkB agonist.
N6272	NPS-2143 Hydrochloride	Ca^{2+}-sensing receptor antagonist.
L9701	LY-450139	GHS-R1a agonist, γ-secretase inhibitor.
J0001	J-147	Phenyl hydrazide; neuronal oxidative stress inhibitor.

Angiogenesis Inhibitors

Angiogenesis is a process in which new blood vessels are formed from old blood vessels; it is responsible for nearly all vessel growth during development and disease. Angiogenesis is a normal, healthy process necessary for development and wound healing but it can also drive the formation of tumors in cancerous growths. Inhibition of angiogenesis prevents tumor growth by limiting the blood and nutrient supply to cancerous tissues. Because of this, angiogenesis makes an excellent target in the development of new chemotherapeutics.

Product ID	Product Name	Description
E8657	Evodiamine	Indole alkaloid found in *Evodia rutaecarpa*; topoisomerase I and II inhibitor.
E5219	Endothelin-1, human	Endogenous peptide involved in vascular contraction. ET-A/B agonist.
P0270	Parthenolide	Sesquiterpene lactone found in *Tanacetum*; TRPA1 partial agonist, NLRP3 and caspase 1 inhibitor.
P1869	Perindopril Erbumine	ACE inhibitor.
T0250	Tamoxifen Ctrate	4-Hydroxytamoxifen prodrug; FIASMA, SERM, PKC inhibitor.
S8253	Sunitinib Malate	Indoline; PDGFR, VEGFR, c-KIT, FLT3, mTORC1 inhibitor.
A4443	L-(+)-Alliin	Optically active cysteine derivative found in *Allium*.
E5221	Endothelin-2, human	Endogenous peptide involved in vascular contraction. ET-A/B agonist.
V0147	Valproic Acid Sodium Salt	T-type Ca^{2+} and voltage-gated Na^{+} channel blocker, GABA transaminase and HDAC inhibitor.
D1627	Dehydrocostus Lactone	Sesquiterpene lactone found in *Saussurea*.
V0376	Vatalanib Dihydrochloride	VEGFR inhibitor.
Z5744	Zoledronic Acid Hydrate	Bisphosphonate; FPPS inhibitor.
E5057	Emodin	Anthraquinone found in various plant sources; CTFR Cl^{-} channel activator.
N5669	Nordihydroguaiaretic Acid	Phenol found in the creosote bush; 5-lipoxygenase, AChR, BChE, mTORC1 inhibitor.
D3202	Diallyl Trisulfide	Organosulfur found in garlic.
S5868	Sorafenib	c-Raf, Ret, VEGFR2 inhibitor, potential STAT3/5 inhibitor, potential HMT inhibitor.
N5550	Nomilin	Triterpene found in species of *Citrus*; HIV-1 protease and aromatase inhibitor.
R3577	Ritonavir	HIV protease inhibitor.
P2513	Phenyl Isothiocyanate	ITC found in cruciferous vegetables.
G8225	Guggulsterone	Steroid found in myrrh; FXR antagonist.
I5213	Indole-3-carbinol	Indole, glucosinolate found in cruciferous vegetables.
T6902	Tranilast	Mast cell stabilizer; TRPV2 antagonist, PDGFR inhibitor.
I6932	Irinotecan	Camptothecin analog; topoisomerase I inhibitor, potential AChE inhibitor.
D3209	Diclofenac Sodium Salt	NSAID; COX-1/2 inhibitor, potential K^{+} channel modulator.
A9435	Axitinib	VEGFR inhibitor.
T0152	Tandutinib	FLT3, PDGFR, c-Kit inhibitor.
G3556	20(S)-Ginsenoside Rg3	Triterpene saponin found in species of *Panax*; γ2 GABA-A agonist, Kv7.1 K^{+} channel activator, α10 nAChR antagonist.
G3552	20(R)-Ginsenoside Rg3	Triterpene saponin found in species of *Panax*; γ2 GABA-A agonist, Kv7.1 K^{+} channel activator, α10 nAChR antagonist.
P0397	Pazopanib	VEGFR, PDGFR, c-Kit inhibitor.
P7358	Psoralen	Furanocoumarin found in *Psoralea coryfolia*, DNA cross-linker; topoisomerase I inhibitor.
V0352	Vandetanib	RET, EGFR, VEGFR2 inhibitor.
A6368	Aprepitant	NK1 antagonist.
F0275	Fasudil	ROCK inhibitor.
F3473	Fisetin	Flavonoid found in various plant sources; matrix metalloproteinase and topoisomerase I and II inhibitor.
T2834	Thiolutin	Dithiolopyrrolone; RNA polymerase inhibitor.
E4444	Ellagic Acid	**Phenol found in various fruits; HDAC modulator.**
P2508	Phenethyl Isothiocyanate	ITC found in cruciferous vegetables.
B1653	Benzyl Isothiocyanate	ITC found in cruciferous vegetables.
L3250	D-Limonene	Terpene found in the rind of various citrus fruits.
C0020	Cafestol	Diterpene found in brewed, unfiltered coffee; FXR and PXR agonist, ERK2 and MEK1 inhibitor.
K0030	Kahweol	Diterpene found in coffee beans.

Product ID	Product Name	Description
P0219	Paeonol	Found in *Paeonia*, *Arisaema*, *Dioscorea*; MAO-A/B inhibitor, voltage-gated and receptor-gated Ca^{2+} channel blocker.
N0069	Naringin	Flavanone glycoside found in citrus fruit; SERM.
C7097	Cryptotanshinone	Quinoid diterpene found in species of *Salvia*; STAT3 inhibitor.
H5654	Honokiol	Lignan found in species of *Magnolia*; GABA-A potentiator.
M0125	Magnolol	Lignan found in species of *Magnolia*; GABA-A potentiator.
D3330	Dihydrotanshinone	Found in *Salvia*; fatty acid synthase and AChE inhibitor, mineralocorticoid and glucocorticoid antagonist.
T0153	Tanshinone I	Diterpene found in *Salvia*.
T7032	Triamcinolone	Steroid; glucocorticoid agonist.
S8098	SU-1498	Tyrphostin; VEGFR inhibitor.
B1979	Betulinic acid	Pentacyclic triterpene.
Z1970	Zerumbone	Cyclic sesquiterpene found in ginger root; potential TRPV1 antagonist.
N9816	2-Hydroxy Estradiol	Estradiol metabolite.
W5727	Wogonoside	Flavonoid glycoside found in *Scutellaria*.
G4400	Glabridin	Isoflavonoid found in *Glycyrrhiza*; GABA-A positive modulator.
I7559	Isoliquiritigenin	Chalcone; SIRT activator, GABA-A positive modulator, NMDA antagonist, VEGFR2 and HDAC inhibitor.
N5986	Novobiocin Sodium Salt	Aminocoumarin; bacterial DNA gyrase inhibitor.
E4418	β-Elemene	Sesquiterpene found in various plant sources.
I7759	Isoxanthohumol	Prenylflavinoid, derivative of xanthohumol found in *Humulus lupulus*.
R1626	Regorafenib Monohydrate	VEGFR1/2/3, TIE2, PDGFRβ, FGFR1, c-Kit, RET, B-Raf inhibitor.
S0400	SB-431542	ALK4/5/7 (activin-like) inhibitor, TGF-β inhibitor.
G7347	GSK-2656157	PERK inhibitor.
T3461	Tiplaxtinin	PAI-1 inhibitor.
T0216	TAE-226	FAK inhibitor.
E4668	ELR-510444	Microtubule polymerization inhibitor.
M3232	Methylisoindigotin	Indirubin derivative.
S4244	SKLB 610	VEGFR2, PDGFR, FGFR2 inhibitor.
X4424	XL-765	PI3K and mTOR inhibitor.
N8660	NVP-AUY922	Isoxazole derivative; HSP90 inhibitor.
B5870	Borrelidin	Macrolide; threonyl-tRNA synthetase/ligase inhibitor.
R3476	RITA	p53 activator.
F5968	Foretinib	ROS1, MET, Ron, Axl, TIE-2, VEGFR2 inhibitor.
B8277	Butein	Flavonoid found in *Rhus verniciflua* and *Butea monosperma*.
I5212	Indirubin	Bisindole isomer of indigo found in *Indigo naturalis*; potential EGFR and CDK inhibitor.
P2002	PF-04691502 Dihydrate	PI3K and mTOR inhibitor.
M4652	MLN8237 (Alisertib)	AurKA inhibitor.
P9200	PX-866	Wortmannin analog; PI3K inhibitor.
P0246	Palomid 529	mTOR inhibitor.
I0800	IC-87114	p110δ PI3K inhibitor.
G1209	GDC-0980	PI3K and mTOR inhibitor.
D3201	Diallyl Sulfide	Organosulfur found in garlic.
A6818	Arenobufagin	Bufanolide steroid found in *Bufo arenarum*;VEGFR2 inhibitor, cardiovascular Na^+/K^+ ATPase inhibitor.
N8460	NVP-BHG712	EphB4 inhibitor.
L1881	Leuprolide Acetate	GnRH analog; GnRH1 agonist.
A4534	Aliskiren Hemifumarate	Renin inhibitor.
L1852	Lenalidomide	Thalidomide derivative; cereblon and TNF-α inhibitor.
E6245	Eplerenone	Mineralocorticoid antagonist.
T3585	Tivozanib	VEGFR1/2/3, c-Kit, PDGFR inhibitor.
C1613	Cediranib	VEGFR inhibitor.
T7044	Triamcinolone Acetonide Acetate	Steroid; glucocorticoid agonist.
M5876	Motesanib	VEGFR1/2/3, PDGFR, c-Kit, RET inhibitor.
V0146	Valsartan	AT1 inhibitor.
P2922	Phenylhexyl Isothiocyanate	ITC found in cruciferous vegetables; HDAC inhibitor.
O4578	Oltipraz	Antioxidant; Nrf2 activator.
T7035	Triptolide	Diterpenoid epoxide found in *Tripterygium*.

Cognitive Enhancers

Product ID	Product Name	Description
C2968	Chrysin	Flavone found in *Passiflora, Oroxylum*, and *Pleurotis*; HDAC2/8 inhibitor, potential aromatase inhibitor.
D1629	Dehydroepiandrosterone	Endogenous steroid hormone; ERβ, NMDA, σ1 agonist, ERα and AR partial agonist, GABA-A antagonist.
S3449	Simvastatin	Statin; HMG-CoA reductase inhibitor, potential ATP-sensitive K^+ channel activator, L-type Ca^{2+} channel blocker.
C0265	Carnosic Acid	Diterpene found in *Rosmarinus*.
A7333	Asiaticoside	Triterpene found in *Centella*, prevents melanogenesis.
T0154	Tanshinone IIA	Natural product found in *Salvia*; MAG lipase inhibitor.
B6998	Bryostatin 1	Macrolide lactone found in *Bugula*; TLR4 activator, PKC modulator.
G3453	Ginsenoside Rh2	Triterpene saponin found in *Panax*.
B3345	(-)-Bilobalide	Sesquiterpene lactone found in *Ginkgo*; GABA-A antagonist.
E6997	Erythropoietin	Endogenous glycoprotein hormone involved in red blood cell production; EpoR agonist.
M3321	Mifepristone	Synthetic steroid, contraceptive; PR and glucocorticoid antagonist.
T1777	S,S-(+)-Tetrandrine	Benzylisoquinoline alkaloid found in *Stephania*; voltage-gated Ca^{2+} channel blocker.
N0069	Naringin	Flavanone glycoside found in *Citrus*; SERM.
G3556	Ginsenoside Rg3	Triterpene saponin found in species of *Panax*; γ2 GABA-A agonist, Kv7.1 K^+ channel activator, α10 nAChR antagonist.
G3557	Ginsenoside Rh1	Triterpene saponin found in species of *Panax*.
P7318	Pseudoginsenoside F11	Saponin found in species of *Panax*; PPARγ agonist.
M0125	Magnolol	Lignan found in *Magnolia*; GABA-A potentiator.
M1749	Memantine Hydrochloride	D2 agonist, NMDA, 5-HT3, α7 nAChR antagonist.
T2816	L-Theanine	Non-essential amino acid found in *Camellia* and *Boletus*, glutamic acid analog; weak AMPA and NMDA agonist.
C1637	Ceftriaxone Sodium	β-lactam cephalosporin; penicillin binding protein inhibitor.
I1418	Idebenone	Synthetic quinone CoQ analog, promotes mitochondrial respiration and ATP production.
N3448	Nimodipine	L-type Ca^{2+} channel blocker.
P7023	Pregnenolone	Endogenous precursor to all steroid hormones; TRPM3 agonist, NMDA potentiator, GABA-A negative allosteric modulator.
S0930	Schisantherin A	Dibenzocyclooctadiene found in *Schisandra*.
A6234	Apigenin	Flavonoid found in various plant sources; GABA-A positive modulator, potential microtubule depolymerization inducer.
C9610	D-Cycloserine	NMDA partial agonist, D-Ala-D-Ala ligase inhibitor.
G2868	Ghrelin, human	Endogenous peptide hormone, involved in feeding behavior and energy homeostasis; ghrelin agonist.
H8162	(-)-Huperzine A	Sesquiterpene alkaloid found in *Huperzia serrata*; AChE inhibitor, NMDA antagonist.
M7528	α-Melanocyte Stimulating Hormone	Endogenous peptide hormone, derived from POMC, involved in energy homeostasis and melanin production; MCR agonist.
N1986	Neuropeptide Y, human/rat	Endogenous peptide neurotransmitter, involved in feeding behavior, stress signaling, circadian rhythm; Y1-5 agonist.
P3465	Piperine	Alkaloid found in black and long peppers; TRPV1 agonist.
D3355	Diosgenin	Steroidal saponin found in *Dioscorea*.
A5477	Antide Acetate	GnRH antagonist.
A6002	Apamin	Peptide, bee venom toxin; SK2/3/4 K^+ channel blocker.
D0033	Daidzin	Isoflavone, phytoestrogen, found in soy.
G0044	Galantide	Peptide; galanin antagonist.
M0035	M35	Peptide; galanin antagonist.
N0160	NAP Peptide	Octapeptide derived from activity-dependent neuroprotective protein (ADNP).
L1784	Levetiracetam	SV2A synaptic vesicle inhibitor.
N1721	Nefiracetam	Pyrrolidone; NMDA and mGluR5 agonist, N-type and L-type Ca^{2+} channel activator, α4β2 nAChR potentiator.
Q8019	Quetiapine Fumarate	5-HT1A and σ1/2 agonist, 5-HT2A/2C/6/7, D1-4, histamine H1/2, M1 mAChR, α1A/1B/2C-adrenergic antagonist.
R3586	Rivastigmine Hydrogen Tartrate	AChE and BChE inhibitor.
A7034	Aripiprazole	D2 and 5-HT1A partial agonist, 5-HT2C/6/7 antagonist, SERT inhibitor.
B5874	Bosutinib	Src and Abl inhibitor.
C4558	Clonidine Hydrochloride	Imidazoline and α2-adrenergic agonist, Nav1.7 Na^+ channel blocker.
F3473	Fisetin	Flavonoid found in various plant sources; matrix metalloproteinase inhibitor, topoisomerase I and II inhibitor.
G3552	20S-Ginsenoside Rg3	Triterpene saponin found in *Panax*; Kv7.1 K^+ channel activator, γ2 GABA-A agonist, α10 nAChR antagonist.
G3558	20R-Ginsenoside Rh2	Triterpene saponin found in *Panax*.
Z3444	Zileuton	5-Lipoxygenase inhibitor.

Product ID	Product Name	Description
T0249	Tamibarotene	RARα/β agonist.
V0369	Varenicline Tartrate	α7 and β4 nAChR agonist, α4β2 and α6β2 nAChR partial agonist.
V3345	Vildagliptin	DPP4 inhibitor.
N3208	Nicardipine	Dihydropyridine; L-type Ca^{2+} channel blocker.
D8014	DU-14	Steroid sulfatase inhibitor.
D5753	Donepezil Hydrochloride	GSK3 and AChE inhibitor, potential σ1 agonist.
I4961	Imperatorin	Furocoumarin; voltage-gated Na^{+} channel blocker, AChE and BChE inhibitor.
G1853	Genipin	Iridoid glycoside found in *Gardinia jasminoides*; AChE inhibitor.
N5605	Nobiletin	Polymethoxylated flavone found in citrus fruits; potential AMPA positive modulator.
B5875	Bosutinib, structural isomer	Bosutinib isomer; Src and Abl inhibitor.
P6819	Presenegenin	Triterpene saponin found in *Polygala*; AChR inhibitor.
G4400	Glabridin	Isoflavonoid found in *Glycyrrhiza*; GABA-A positive modulator.
L5624	Loganin	Iridoid glucoside found in *Cornus officialis*; β-secretase inhibitor.
V5870	Vortioxetine	5-HT1A agonist, 5-HT1B partial agonist, 5-HT3A/7 antagonist, SERT inhibitor, potential β1-adrenergic agonist.
P0109	P7C3A20	Fluorinated aminopropyl carbazole, neuroprotective.
S1855	Senktide Trifluoroacetate Salt	NK3 agonist.
P0013	P7C3	Aminopropyl carbazole, neuroprotective.
S1609	Securinine	Alkaloid found in *Securinega*; potential GABA-A antagonist.
G0246	Galantamine Hydrobromide	Alkaloid found in *Galanthus, Narcissus, Leucojum*, and *Lycoris*; AChE inhibitor, α7 nAChR agonist, mAChR agonist.
P1869	Perindopril Erbumine	ACE inhibitor.
S8005	Substance P	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8006	Substance P (1-4)	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8007	Substance P (1-7)	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8008	Substance P (1-9)	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8009	Substance P (7-11)	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8010	[Nle11]-Substance P	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8012	[Sar9]-Substance P	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8013	[Tyr8]-Substance P	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
S8014	Substance P, Free Acid	Endogenous tachykinin peptide, involved in inflammation, stress signaling, nociception; NK1 agonist.
D3329	7,8-Dihydroxyflavone Hydrate	TrkB agonist.
A5061	Ampalex	Benzylpiperidine; AMPA potentiator.

Insulin Signaling

Insulin is a peptide hormone produced by islet β cells in the pancreas; it is involved in the metabolism of carbohydrates and fats. Insulin promotes absorption of excess glucose from blood to muscle and fat, where it is stored. Insulin also inhibits hepatic glucose production. Dysregulation in insulin signaling can cause hypoglycemia, diabetes mellitus, and metabolic syndrome. Metabolic syndrome and type 2 diabetes mellitus can be caused by obesity or inactivity. Type 1 diabetes mellitus is typically characterized as an idiopathic autoimmune disease involving destruction of insulin-producing β cells. Treatments for diabetes mellitus include compounds that mimic the structure of insulin, decrease production of glucose, increase uptake of glucose, slow conversion of carbohydrates into sugars, or increase insulin release or insulin sensitivity. α-Glucosidase and α-amylase inhibitors prevent or slow the digestion of carbohydrates, limiting blood glucose levels. Glucagon-like peptide 1 (GLP-1) agonists activate GLP-1 receptors to increase insulin synthesis and release. ATP-sensitive K^+ channel blockers induce depolarization of islet cells and stimulate insulin release. Activators of peroxisome proliferator-activated receptor PPAR-γ influence transcription of insulin-sensitive genes, increasing production of insulin-dependent enzymes that increase glucose uptake. Other compounds that alter insulin signaling or glucose levels prevent GLP-1 degradation by dipeptidyl peptidase 4 (DPP4), reduce hepatic glucose output by activating AMPK signaling, and increase insulin synthesis by stimulating β cell proliferation.

α-Glucosidase Inhibitors

Product ID	Product Name	Description
A0802	Acarbose	α-Glucosidase inhibitor.
C0121	Caffeic Acid	Hydroxycinnamic acid found in coffee, argan oil, *Eucaplyptus, Salvinia*, and *Phellinus*; α-amylase and α-glucosidase inhibitor.
E6234	Epigallocatechin Gallate	Flavanol found in *Camilla sinensis* (green tea); AhR antagonist, STAT3, α-amylase, α-glucosidase inhibitor.
M1560	Methyl Caffeate	Polyphenol found in species of *Solanum* and *Magnolia*; α-glucosidase inhibitor.
V5725	Voglibose	α-Glucosidase inhibitor, potential GLP-1 agonist, potential ATP-sensitive K+ channel activator.

α-Amylase Inhibitors

Product ID	Product Name	Description
B3573	Bisdemethoxycurcumin	Curcumin derivative; DNMT1 and α-amylase inhibitor, WIF-1 promoter demethylation inhibitor, potential SIRT1 and AMPK activator.
C0121	Caffeic Acid	Hydroxycinnamic acid found in coffee, argan oil, *Eucaplyptus, Salvinia*, and *Phellinus*; α-amylase and α-glucosidase inhibitor.
E6234	Epigallocatechin Gallate	Flavanol found in *Camilla sinensis* (green tea); AhR antagonist, STAT3, α-amylase, α-glucosidase inhibitor.
G0243	(-)-Gallocatechin	Polyphenol found in *Camilla sinensis*; HIV integrase and RT inhibitor, α-amylase inhibitor.

GLP-1 Agonists

Product ID	Product Name	Description
E9416	Exendin-3	Peptide found in *Heloderma*; GLP-1 agonist.
E9418	Exendin 3 (9-39)	Peptide found in *Heloderma*; GLP-1 agonist.
E9417	Exendin-4 (Exenatide)	Peptide found in *Heloderma*; GLP-1 agonist.
G4481	Glucagon-Like Peptide I Amide (7-36), human	Endogenous peptide hormone, GLP-1 fragment, involved in insulin secretion and feeding behavior; GLP1 agonist.
G4482	Glucagon-Like Peptide I (7-37)	Endogenous peptide hormone, GLP-1 fragment, involved in insulin secretion and feeding behavior; GLP1 agonist.
V5725	Voglibose	α-Glucosidase inhibitor, potential GLP-1 agonist, potential ATP-sensitive K+ channel activator.

ATP-sensitive K^+ Channel Blockers

Product ID	Product Name	Description
E2002	Efaroxan Hydrochloride	α2-Adrenergic and imidazoline-1 antagonist, ATP-sensitive K^+ channel blocker.
G4532	Gliclazide	Sulfonylurea; SUR1 antagonist.
G4535	Glimepiride	Sulfonylurea; ATP-sensitive K^+ channel blocker.
G4634	Glipizide	Sulfonylurea; ATP-sensitive K^+ channel blocker.
M3577	Mitiglinide Calcium	ATP-sensitive K^+ channel blocker, potential RyR agonist.
P3461	Pipemidic Acid	Fluoroquinolone; bacterial DNA gyrase inhibitor and ATP-sensitive K^+ channel blocker.
R1860	Repaglinide	Sulfonylurea; ATP-sensitive K^+ channel blocker.
T7056	Troglitazone	Thiazolidinedione; PPARγ agonist, ATP-sensitive K^+ channel blocker.
V5725	Voglibose	α-Glucosidase inhibitor, potential GLP-1 agonist, potential ATP-sensitive K^+ channel activator.

PPAR-γ Activators

Product ID	Product Name	Description
C3210	Ciglitazone	Thiazolidinedione; PPARγ agonist.
G3457	Ginsenoside Re	Triterpene saponin found in species of *Panax*; PPARγ agonist.
I6804	Irbesartan	PPARγ agonist, AT1 antagonist.
P6954	Pioglitazone Hydrochloride	Thiazolidinedione; PPARα/γ agonist, mitoNEET modulator.
P7318	Pseudoginsenoside F11	Saponin found in species of *Panax*; PPARγ agonist.
R5773	Rosiglitazone	Thiazolidinedione; PPARγ agonist.
R5772	Rosiglitazone Maleate	Thiazolidinedione; PPARγ agonist.
T1644	Telmisartan	AT1 antagonist, PPARγ/δ modulator.
T7056	Troglitazone	Thiazolidinedione; PPARγ agonist, ATP-sensitive K+ channel blocker.
Z4552	ZLN005	PPARγ coactivator-1α expression stimulator.

Other Insulin Signaling Modulators

Product ID	Product Name	Description
B8676	BVT-2733	11β-HSD1 inhibitor.
C0824	CCG1423	Serum response factor inhibitor and MRTF-A binding agent.
C2965	Chromium Picolinate	Used to prevent chromium deficiency and to decrease serum glucose and insulin levels.
D1773	Deshydroxy LY-411575	γ-Secretase inhibitor.
G1650	Geniposide	Iridoid glycoside found in *Gardenia*; GLUT2 expression stimulator.
H9613	N-(4-Hydroxyphenyl)retinamide (Fenretinide)	Retinol (vitamin A) analog that binds RBP4; Des1 inhibitor.
M2076	Metformin Hydrlochloride	AMPK activator.
M1579	Methazolamide	Carbonic anhydrase inhibitor.
S3476	Sitagliptin Phosphate Monohydrate	DPP4 inhibitor.
S6800	SR1001	RORα/γ inverse agonist.
V1869	Verbascoside	Phenylpropanoid found in *Castilleja, Verbena*, and *Verbascum*; potential PKC, DPP4, prolyl oligopeptidase inhibitor.
V3345	Vildagliptin	DPP4 inhibitor.
W7200	WS3	Islet β cell proliferation stimulator.
W7201	WS6	Islet α/β cell proliferation stimulator.

Parkinson's Disease

Product ID	Product Name	Description
A4544	Allyl disulfide	Organosulfur found in garlic.
C2944	Chlorogenic Acid (from Lonicera)	Polyphenol derivative of caffeic acid found in *Lonicera*; DNMT and G6P translocase inhibitor, potential pepsin inhibitor.
E6880	Erucin	ITC found in cruciferous vegetables, analog of sulforaphane; telomerase inhibitor.
N0068	Naringenin	Flavonone found in species of *Citrus*.
P3269	Piroxicam	NSAID; COX-1/2 inhibitor.
R1777	9-cis-Retinoic Acid	Vitamin A derivative; RAR and RXR agonist.
E6234	Epigallocatechin gallate	Flavanol found in *Camilla* (green tea); AhR antagonist, STAT3, α-amylase, α-glucosidase inhibitor.
S3449	Simvastatin	Statin; HMG-CoA reductase inhibitor, potential ATP-sensitive K^+ channel activator, L-type Ca^{2+} channel blocker.
M1644	Meloxicam	NSAID; COX-2 inhibitor.
S3345	Silymarin	Flavonolignan mixture found in *Silybum* (milk thistle) seeds.
D1792	Dextromethorphan Hydrobromide Hydrate	σ1/2and μ/κ/δ-OR agonist, α3β4/α4β2/α7 nAChR, SERT, NET, NMDA antagonist.
E7309	Esculetin	Coumarin lactone found in chicory and other plant sources; β-catenin inhibitor.
P7318	Pseudoginsenoside F11	Saponin found in species of *Panax*; PPARγ agonist.
M1749	Memantine Hydrochloride	D2 agonist, NMDA, 5-HT3, α7 nAChR antagonist.
A4802	Amantadine Hydrochloride	Viral M2 proton channel blocker, MAO-A, NET, NMDA, α7 nAChR antagonist.
A4803	Amantadine Sulfate	Viral M2 proton channel blocker, MAO-A, NET, NMDA, α7 nAChR antagonist.
L1782	Levodopa	Endogenous catecholamine precursor, also found in *Mucuna*.
K1655	Kenpaullone	GSK-3β, HGK, and CDK inhibitor.
P3465	Piperine	Alkaloid found in black and long peppers; TRPV1 agonist.
R5661	Ropinirole Hydrochloride	Indoline; D2/3 agonist.
Z5653	Zonisamide	Sulfonamide; carbonic anhydrase inhibitor, voltage-gated Na^+ and T-type Ca^{2+} channel blocker.
P6901	Pramipexole Dihydrochloride	D2/3 agonist.
C2943	Chlorogenic Acid (from Eucommia)	Derivative of caffeic acid found in *Eucommia* bark; DNMT and G6P translocase inhibitor, potential pepsin inhibitor.
R0272	Rasagiline Mesylate	MAO-A/B inhibitor.
E5575	Entacapone	COMT inhibitor.
G4781	Glucoraphanin Potassium Salt	Glucosinolate found in cruciferous vegetables.
P0013	P7C3	Aminopropyl carbazole, neuroprotective.
B1992	Bexarotene	RXR agonist.
E0929	Echinacoside	Phenylethanoid glycoside found in *Echinacea*.
M1564	S-Methyl-L-cysteine	Antioxidant found in *Brassicaceae* family plants.
G8850	GW-5074	c-Raf inhibitor.
E2002	Efaroxan Hydrochloride	α2-adrenergic and imidazoline-1 antagonist, ATP-sensitive K^+ channel blocker.
Y1000	Y27632 Dihydrochloride	ROCK inhibitor.
T1978	Tetrahydroberberine	Isoquinoline alkaloid found in *Corydalis*; 5-HT1A agonist, D2 antagonist, ATP-sensitive K^+ channel blocker.
I7478	Istradefylline	Adenosine A2A antagonist.
S7819	Stepholidine	Alkaloid found in *Stephania intermedia*; D1 agonist, 5-HT1A partial agonist, D2 antagonist.
P0109	P7C3A20	Fluorinated aminopropyl carbazole, neuroprotective.
P0110	(R)-P7C3-OMe	Methoxy aminopropyl carbazole, neuroprotective.
F4532	Flibanserin	5-HT1A agonist, 5-HT2A antagonist.
M0113	Madecassoside	Triterpenoid found in *Centella*.
D3349	Dimebon Dihydrochloride	AMPK activator, L-type Ca^{2+} channel and NMDA, histamine H1/2, α-adrenergic, 5-HT2C/5A/6 receptor antagonist.
G5216	GNE-7915	LRRK2 inhibitor.
D3329	7,8-Dihydroxyflavone Hydrate	TrkB agonist.
R5700	Ro 61-8048	Kynurenine 3-hydroxylase inhibitor.
C9808	CZC-54252	LRRK2 inhibitor.
P2100	PF-06447475	LRRK2 inhibitor.

Ca^{2+} Channel Modulators

Product ID	Product Name	Description
H1672	Hesperetin	Flavanone found in species of *Citrus*; L-type Ca^{2+} channel blocker.
N3208	Nicardipine	Dihydropyridine; L-type Ca^{2+} channel blocker.
N1721	Nefiracetam	Pyrrolidone; NMDA and mGluR5 agonist, N-type and L-type Ca^{2+} channel activator, α4β2 nAChR potentiator.
T1777	S,S-(+)-Tetrandrine	Benzylisoquinoline alkaloid found in *Stephania*; voltage-gated Ca^{2+} channel blocker.
N3322	Niflumic Acid	NSAID; NMDA inverse agonist, T-type Ca^{2+} and Cl^- channel blocker, GABA-A antagonist, COX-1/2 inhibitor.
T1678	D,L-Tetrahydropalmatine	Alkaloid found in *Corydalis* and *Stephania*; D1/2 antagonist, potential L-type Ca^{2+} and K^+ channel blocker.
R3197	Rhyncholphylline	Oxyindole alkaloid found in *Uncaria sinensis*; hERG K^+ and L-type Ca^{2+} channel channel blocker, NMDA antagonist.
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^+ channel blocker, GLT-1 modulator.
V0147	Valproic Acid Sodium Salt	T-type Ca^{2+} and voltage-gated Na^+ channel blocker, GABA transaminase and HDAC inhibitor.
L5751	Lomerizine Dihydrochloride	L-type and T-type Ca^{2+} and TRP antagonist.
A4440	Allicin	Organosulfur found in garlic, binds DNA; inwardly rectifying K^+ channel activator, L-type Ca^{2+} channel blocker.
A4441	Allicin, aqueous	Organosulfur found in garlic, binds DNA; inwardly rectifying K^+ channel activator, L-type Ca^{2+} channel blocker.
T6934	Trimebutine Maleate	L-type Ca^{2+} channel blocker, BK K^+ channel modulator, potential opioid agonist.
P0218	Paeoniflorin	Found in *Paeonia*; L-type Ca^{2+} channel blocker.
V1769	Verapamil Hydrochloride	L-type Ca^{2+} channel blocker.
H9861	Hypericin	Naphthodianthrone found in *Hypericum*; dopamine β-hydroxylase and proteasome inhibitor, N-type and P/Q-type Ca^{2+} channel blocker.
A5315	Deoxy-andrographolide	Labdane diterpene found in *Andrographolis*; cNOS and adenylyl cyclase activator, PAF inhibitor, potential voltage-gated Ca^{2+} channel blocker.
R5774	Roscovitine	CDK inhibitor, L-type Ca^{2+} channel blocker.
P0005	Pituitary Adenylate Cyclase-activating Polypeptide (1-27), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0006	Pituitary Adenylate Cyclase-activating Polypeptide (1-38), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0007	Pituitary Adenylate Cyclase-activating Polypeptide (6-27), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0009	Pituitary Adenylate Cyclase-activating Polypeptide (1-38), frog	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0010	Pituitary Adenylate Cyclase-activating Polypeptide-related Peptide, human	Peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0011	Pituitary Adenylate Cyclase-activating Polypeptide-related Peptide, rat	Peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0008	Pituitary Adenylate Cyclase-activating Peptide (6-38), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
S5749	Somatostatin-14	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
N3228	Nifedipine	Dihydropyridine; L-type Ca^{2+} channel blocker.
D3227	Dihydromethysticin	Kavalactone found in *Piper methysticum* (kava plant); Voltage-gated Na^+ and L-type Ca^{2+} channel blocker.
K0088	Kawain	Kavalactone found in *Piper methysticum* (kava plant); Voltage-gated Na^+ and L-type Ca^{2+} channel blocker.
A5037	Amiodarone Hydrochloride	Voltage-gated Na^+, Ca^{2+}, K^+ channel blocker, α/β-adrenergic antagonist, FIASMA.
Z5653	Zonisamide	Sulfonamide; carbonic anhydrase inhibitor, voltage-gated Na^+ and T-type Ca^{2+} channel blocker.
L0349	Lamotrigine	R-type Ca^{2+} and voltage-gated Na^+ channel blocker.
P0219	Paeonol	Found in *Paeonia, Arisaema*, and *Dioscorea*; MAO-A/B inhibitor, voltage-gated and receptor-gated Ca^{2+} channel blocker.
P7103	Praziquantel	Alters membrane permeability and Ca^{2+} signaling; potential adenosine antagonist, potential voltage-gated Ca^{2+} channel blocker.
C0376	Catharanthine base	Alkaloid found in *Catharanthus*, precursor in synthesis of vinca alkaloids; voltage-gated Ca^{2+} channel blocker.
C0377	Catharanthine sulfate	Alkaloid found in *Catharanthus*, precursor in synthesis of vinca alkaloids; voltage-gated Ca^{2+} channel blocker.
C0378	Catharanthine Tartrate	Alkaloid found in *Catharanthus*, precursor in synthesis of vinca alkaloids; voltage-gated Ca^{2+} channel blocker.
P0253	Panaxadiol	Triterpene sapongenin found in species of *Panax*; voltage-gated Ca^{2+} channel blocker.
P0254	Panaxatriol	Triterpene sapogenin found in species of *Panax*; voltage-gated Ca^{2+} channel blocker.
SC7056	Snake venom - Crotalus durissus terrificus	Snake venom found in *Crotalus durrisus terrificus*; binds N-type and P/Q-type Ca^{2+} channels, L-type Ca^{2+} channel potentiator, NMJ blocker.
A5045	Amlodipine	Dihydropyridine, FIASMA; L-type Ca^{2+} channel blocker.

Product ID	Product Name	Description
A5044	Amlodipine besylate	Dihydropyridine, FIASMA; L-type Ca^{2+} channel blocker.
G0106	Gabapentin	GABA analog; GABA potentiator, adenosine A1 agonist, voltage-gated α2δ Ca^{2+} channel blocker, NMDA modulator.
M0262	Maprotiline Hydrochloride	Tetracycline; FIASMA, histamine H1, 5-HT2, mAChR, α1-adrenergic antagonist, L-type Ca^{2+} channel blocker, NET inhibitor.
N3448	Nimodipine	L-type Ca^{2+} channel blocker.
C3251	Cinnarizine	FIASMA, L-type Ca^{2+} channel blocker, D2 antagonist.
S5750	Somatostatin-28	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
S5745	[Tyr1]-Somatostatin	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
S5747	[Tyr11]-Somatostatin	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
S5751	Somatostatin-25	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
S5752	Somatostatin-28 (1-12)	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
S5753	Somatostatin-28 (1-14)	Endogenous neuropeptide hormone; somatostatin agonist, GABA modulator, P/Q-type, N-type, and L-type voltage-gated Ca^{2+} channel modulator.
T6935	Trimebutine base	BK K^+ channel and L-type Ca^{2+} channel blocker.
C0247	Calcineurin Autoinhibitory Peptide	Peptide; calcineurin inhibitor.
F1745	Felodipine	Dihydropyridine; L-type Ca^{2+} channel blocker.
G4483	Glucagon-Like Peptide II, human	Endogenous peptide hormone, involved in intestinal function; GLP2 agonist.
G4484	Glucagon-Like Peptide II, rat	Endogenous peptide hormone, involved in intestinal function; GLP2 agonist.
G4485	[Ala19]-Glucagon-Like Peptide II, rat	Peptide hormone, GLP-2 derivative; GLP2 agonist.
P1854	Penicillic Acid	Neuroactive mycotoxin produced by *Penicillum* and *Aspergillus*; BK K^+, voltage-gated cardiac Na^+, K^+, Ca^{2+} channel inhibitor.
C3446	Cilnidipine	Dihydropyridine; L-type and N-type Ca^{2+} channel blocker.
A5235	Amitriptyline Hydrochloride	FIASMA, σ1, RyR2, TrkA/B agonist, 5-HT2/6/7, M1-5 mAChR, histamine H1/4, α1-adr. antagonist, SERT, NET inhibitor, Kv1.1/7.2/7.3 K^+, L-type Ca^{2+} channel blocker.
C9863	Cyproconazole	Triazole; 14-α demethylase inhibitor, voltage-gated Ca^{2+} channel blocker, potential aromatase inhibitor.
T1605	Tebuconazole	Triazole; 14-α demethylase inhibitor, voltage-gated Ca^{2+} channel blocker, potential aromatase inhibitor.
M0248	Manidipine Hydrochloride	Dihydropyridine; L-type and T-type Ca^{2+} channel blocker.
D3447	Diltiazem Hydrochloride	Benzothiazepine; L-type Ca^{2+} channel blocker, potential CNG channel blocker.
I7258	Isoflurane	Ca^{2+} ATPase and ATP-sensitive K^+ channel activator, GABA-A and GlyR potentiator, NMDA antagonist, L-type Ca^{2+}, delayed rectifier K^+, A-type K^+ channel blocker.
P7158	Protopine	Isoquinoline alkaloid found in a variety of plant sources; voltage- and receptor-gated Ca^{2+} channel blocker.
C4457	Clomipramine Hydrochloride	FIASMA, mAChR, 5-HT2/3/6/7, α1/2-adrenergic antagonist, SERT and NET inhibitor, hERG K^+ and L-type Ca^{2+} channel blocker.
D3349	Dimebon Dihydrochloride	AMPK activator, L-type Ca^{2+} channel and NMDA, histamine H1/2, α-adrenergic, 5-HT2C/5A/6 receptor antagonist.

K^+ Channel Modulators

Product ID	Product Name	Description
D3209	Diclofenac Sodium Salt	NSAID; COX-1/2 inhibitor, potential K^+ channel modulator.
G4597	18β-Glycyrrhetinic Acid	Triterpene glycoside found in *Glycyrrhiza*; 15-HPGDH inhibitor, hERG and KCNA3/Kv1.3 K^+ channel blocker.
A4440	Allicin	Organosulfur found in garlic, binds DNA; inwardly rectifying K^+ channel activator, L-type Ca^{2+} channel blocker.
P6852	Propafenone Hydrochloride	β-adrenergic antagonist, Kv1.4 and K2P2 K^+ channel blocker.
P2817	Phentolamine Hydrochloride	ATP-sensitive K^+ channel activator, α-adrenergic antagonist.
P2818	Phentolamine Methanesulfonate	ATP-sensitive K^+ channel activator, α-adrenergic antagonist.
T7056	Troglitazone	Thiazolidinedione; PPARγ agonist, ATP-sensitive K^+ channel blocker.
G3556	Ginsenoside Rg3	Triterpene saponin found in species of *Panax*; γ2 GABA-A agonist, Kv7.1 K^+ channel activator, α10 nAChR antagonist.
P6958	Protopanaxatriol	Triterpene sapogenin found in species of *Panax*; GABA-A/C antagonist, slow-activating delayed rectifier K^+ channel blocker.
V3355	Vindoline	Semi-synthetic vinca alkaloid found in *Catharanthus*; Kv2.1 K^+ channel blocker and H^+/K^+ ATPase inhibitor.
A5037	Amiodarone Hydrochloride	Voltage-gated Na^+, Ca^{2+}, K^+ channel blocker, α/β-adrenergic antagonist, FIASMA.
B8262	Bupivacaine Hydrochloride Monohydrate	Amino amide; voltage-gated Na^+, BK/SK, Kv1, Kv3, TASK-2 K^+ channel inhibitor.
C0270	Carbamazepine	GABA potentiator, voltage-gated Na^+ and ATP-sensitive K^+ channel blocker.
C9711	Cyclovirobuxine D	Found in *Buxus*; hERG K^+ channel inhibitor.
D5649	Domperidone	D2/3 antagonist, hERG K^+ channel blocker.
G4535	Glimepiride	Sulfonylurea; ATP-sensitive K^+ channel blocker.
G4634	Glipizide	Sulfonylurea; ATP-sensitive K^+ channel blocker.
I5034	Imiquimod	Imidazoquinoline nucleoside analog; TLR-7/8 agonist, KCNA1/Kv1.1 and KCNA2/Kv1.2 K^+ channel partial agonist, TREK-1/K2P2 and TRAAK/K2P4 K^+ channel blocker.
N1822	Nefazodone Hydrochloride	5-HT2 antagonist, SERT and NET inhibitor, hERG K^+ channel blocker.
O9210	Oxcarbazepine	α4β2 nAChR desensitizer, delayed-rectifier voltage-gated K^+ and voltage-gated Na^+ channel blocker.
B5648	Bombesin	GRP analog found in *Bombina*; bombesin and GRP agonist, hERG K^+ channel blocker.
F4584	Fluphenazine Hydrochloride	Piperazine; FIASMA, D2 antagonist, hERG K^+ channel blocker.
L1884	Levosimendan	Ca^{2+} sensitizer; ATP-sensitive K^+ channel activator, troponin C stabilizer, PDE inhibitor.
P0008	Pituitary Adenylate Cyclase-activating Peptide (6-38), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
A6002	Apamin	Peptide, bee venom toxin; SK2/3/4 K^+ channel blocker.
C7997	C-Type Natriuretic Peptide (1-22), human	Endogenous cardiomodulatory peptide; NPR-B agonist.
C7998	C-Type Natriuretic Peptide, chicken	Endogenous cardiomodulatory peptide; NPR-B agonist.
D3429	Dihydrocytochalasin B	Mycotoxin derivative; actin polymerization inhibitor, Kv1.1-A-type K^+ channel converter.
N5211	Nocistatin	Neuropeptide; TRPC agonist, BK/SK K^+ channel modulator.
P0005	Pituitary Adenylate Cyclase-activating Polypeptide (1-27), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0006	Pituitary Adenylate Cyclase-activating Polypeptide (1-38), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0007	Pituitary Adenylate Cyclase-activating Polypeptide (6-27), human/sheep/rat	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0009	Pituitary Adenylate Cyclase-activating Polypeptide (1-38), frog	Endogenous peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0010	Pituitary Adenylate Cyclase-activating Polypeptide-related Peptide, human	Peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
P0011	Pituitary Adenylate Cyclase-activating Polypeptide-related Peptide, rat	Peptide, involved in paracrine and autocrine cell signaling; PAC1 and VPAC1/2 agonist.
R3477	Ritodrine Hydrochloride	Phenylethylamine; β2-adrenergic agonist, SK/BK and ATP-sensitive K^+ channel activator.
S0171	Sarafotoxin 6c	Isoform of a toxin found in *Atractaspis engaddensis*; ET-B agonist, ATP-sensitive K^+ channel activator.
S6019	Speract	Peptide, derived from egg outer envelope; K^+ channel activator, speract SRCR agonist.
A5235	Amitriptyline Hydrochloride	FIASMA, σ1, RyR2, TrkA/B agonist, 5-HT2/6/7, M1-5 mAChR, histamine H1/4, α1-adrenergic antagonist, SERT, NET inhibitor, Kv1.1/7.2/7.3 K^+, L-type Ca^{2+} channel blocker.
A5059	Amoxapine	5-HT2/3/6/7, D2/3/4, histamine H1, α1-adrenergic antagonist, SERT and NET inhibitor, hERG K^+ channel blocker.
S5976	Sotalol Hydrochloride	β-adrenergic antagonist, voltage-gated Na^+ and K^+ channel blocker.
B8261	Bupivacaine	BK/SK, Kv1, Kv3, TASK-2 K^+ channel blocker, voltage-gated Na^+ channel blocker.
A5072	Amsacrine	Acridine derivative; DNA intercalator, topoisomerase II inhibitor, hERG K^+ channel blocker.

Product ID	Product Name	Description
R1860	Repaglinide	Sulfonylurea; ATP-sensitive K^+ channel blocker.
M3577	Mitiglinide Calcium	ATP-sensitive K^+ channel blocker, potential RyR agonist.
M5727	Moguisteine	Potential ATP-sensitive K^+ channel blocker, potential irritant receptor antagonist.
F4583	Flupirtine Maleate	Kv7 K^+ channel activator, NMDA antagonist, GABA-A negative modulator.
G3552	20S-Ginsenoside Rg3	Triterpene saponin found in *Panax*; Kv7.1 K^+ channel activator, γ2 GABA-A agonist, α10 nAChR antagonist.
D5992	Doxapram Hydrochloride Hydrate	K^+ channel blocker, catecholamine release stimulator.
T2936	Thioridazine Hydrochloride	Piperadine phenothiazine; FIASMA, D1-5, histamine H1/2, M1-5 mAChR, 5-HT1/2/5/6/7, α1/2-adrenergic antagonist, NET inhibitor, hERG K^+ channel inhibitor.
P3461	Pipemidic Acid	Fluoroquinolone; bacterial DNA gyrase inhibitor and ATP-sensitive K^+ channel blocker.
A4441	Allicin, aqueous	Organosulfur found in garlic, binds DNA; inwardly rectifying K^+ channel activator, L-type Ca^{2+} channel blocker.
R3197	Rhyncholphylline	Oxyindole alkaloid found in *Uncaria sinensis*; hERG K^+ and L-type Ca^{2+} channel channel blocker, NMDA antagonist.
C4457	Clomipramine Hydrochloride	FIASMA, mAChR, 5-HT2/3/6/7, α1/2-adrenergic antagonist, SERT and NET inhibitor, hERG K^+ and L-type Ca^{2+} channel blocker.
S1863	Seproxetine Hydrochloride	Fluoxetine metabolite; SERT and DAT inhibitor, Kv7.1 K^+ channel blocker, 5-HT2A/2C antagonist.
T7003	Trazodone Hydrochloride	5-HT1A partial agonist, 5-HT2, histamine, α1/2-adrenergic antagonist, SERT inhibitor, voltage-gated K^+ channel blocker.
I7258	Isoflurane	Ca^{2+} ATPase and ATP-sensitive K^+ channel activator, GABA-A and GlyR potentiator, NMDA antagonist, L-type Ca^{2+}, delayed rectifier K^+, A-type K^+ channel blocker.
G3252	6-Gingerol	Phenol found in *Zingiber*; TRPV1 agonist, 5-HT3 antagonist, TASK-1/TASK-3/TRESK K^+ channel blocker.
F4483	Flufenamic Acid	NSAID; TREK1 K^+ potentiator, voltage-gated Na^+ channel blocker, TRPC3 and TRPM2 antagonist, COX-1/2 inhibitor.
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^+ channel blocker, GLT-1 modulator.
T6934	Trimebutine Maleate	L-type Ca^{2+} channel blocker, BK K^+ channel modulator, potential opioid agonist.
T6935	Trimebutine base	BK K^+ channel and L-type Ca^{2+} channel blocker.
P1854	Penicillic Acid	Neuroactive mycotoxin produced by *Penicillum* and *Aspergillus*; BK K^+, voltage-gated cardiac Na^+, K^+, Ca^{2+} channel inhibitor.
T1978	Tetrahydroberberine	Isoquinoline alkaloid found in *Corydalis*; 5-HT1A agonist, D2 antagonist, ATP-sensitive K^+ channel blocker.

Na^+ Channel Modulators

Product ID	Product Name	Description
A5133	Amiloride Hydrochloride Dihydrate	K^+-sparing diuretic; ENaC and acid-sensing ion channel blocker, Na^+/H^+ antiporter inhibitor.
D3462	Diphenhydramine Hydrochloride	Voltage-gated Na^+ channel blocker, mAChR antagonist, histamine H1 inverse agonist.
T7033	Trifluoperazine Hydrochloride	Phenothiazine; D1/2 and α1-adrenergic antagonist, calmodulin and CDPK4 inhibitor, Nav1.4 and Nav1.7 Na^+ channel blocker.
D3227	Dihydromethysticin	Kavalactone found in *Piper methysticum* (kava plant); Voltage-gated Na^+ and L-type Ca^{2+} channel blocker.
K0088	Kawain	Kavalactone found in *Piper methysticum* (kava plant); Voltage-gated Na^+ and L-type Ca^{2+} channel blocker.
M1679	Methysticin	Kavalactone found in *Piper methysticum* (kava plant).
L0060	Lappaconitine	Alkaloid found in species of *Aconitum*; cardiac Na^+ channel blocker.
B8144	Bulleyaconitine A	Natural product found in *Aconitum bulleyanum*; voltage-gated Na^+ channel blocker.
F4483	Flufenamic Acid	NSAID; TREK1 K^+ potentiator, voltage-gated Na^+ channel blocker, TRPC3 and TRPM2 antagonist, COX-1/2 inhibitor.
A0958	Aconitine	Toxin found in *Aconitum*; voltage-gated Na^+ channel modulator.
A5037	Amiodarone Hydrochloride	Voltage-gated Na^+, Ca^{2+}, K^+ channel blocker, α/β-adrenergic antagonist, FIASMA.
B8262	Bupivacaine Hydrochloride Monohydrate	Amino amide; voltage-gated Na^+, BK/SK, Kv1, Kv3, TASK-2 K^+ channel inhibitor.
C0270	Carbamazepine	GABA potentiator, voltage-gated Na^+ and ATP-sensitive K^+ channel blocker.
B8261	Bupivacaine	BK/SK, Kv1, Kv3, TASK-2 K^+ channel blocker, voltage-gated Na^+ channel blocker.
O9210	Oxcarbazepine	α4β2 nAChR desensitizer, delayed-rectifier voltage-gated K^+ and voltage-gated Na^+ channel blocker.
S6235	Spironolactone	Mineralocorticoid, aldosterone, AR antagonist, ENaC channel blocker, potential voltage-gated Ca^{2+} channel blocker.
H9759	Hypaconitine	Diterpene alkaloid found in *Aconitum*; voltage-gated Na^+ channel modulator.
Z5653	Zonisamide	Sulfonamide; carbonic anhydrase inhibitor, voltage-gated Na^+ and T-type Ca^{2+} channel blocker.
L0349	Lamotrigine	R-type Ca^{2+} and voltage-gated Na^+ channel blocker.
T1674	Terbutaline	β2-adrenergic agonist, potential ENaC activator.
V0147	Valproic Acid Sodium Salt	T-type Ca^{2+} and voltage-gated Na^+ channel blocker, GABA transaminase and HDAC inhibitor.
P7059	Proxymetacaine Hydrochloride	Potential voltage-gated Na^+ channel blocker.
C4558	Clonidine Hydrochloride	Imidazoline and α2-adrenergic agonist, Nav1.7 Na^+ channel blocker.
B6918	Brevetoxin 3	Polyether neurotoxin found in *Karenia brevis*; Nav1.4/1.5 Na^+ channel activator.
B6917	Brevetoxin 2	Polyether neurotoxin found in *Karenia brevis*; Nav1.4/1.5 Na^+ channel activator.
I4961	Imperatorin	Furocoumarin; voltage-gated Na^+ channel blocker, AChE and BChE inhibitor.
D8145	Duloxetine Hydrochloride	SERT and NET inhibitor, Nav1.7 Na^+ channel blocker.
P6870	Propofol	GABA-A agonist, NMDA antagonist, voltage-gated Na^+ channel blocker.
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^+ channel blocker, GLT-1 modulator.
P1854	Penicillic Acid	Neuroactive mycotoxin produced by *Penicillum* and *Aspergillus*; BK K^+, voltage-gated cardiac Na^+, K^+, Ca^{2+} channel inhibitor.
G7200	GS-967	Na^+ channel blocker.
A0001	A-803467	Nav1.8 Na^+ channel blocker, potential Nav1.5 Na^+ channel blocker.

TRP Modifiers

Product ID	Product Name	Description
A0817	D,L-1'-Acetoxychavicol Acetate	Found in *Languas* and *Alpinia*; TRPA1 agonist, xanthine oxidase inhibitor.
C0368	Carveol	Terpene alcohol found in spearmint oil; TRPV3 agonist.
R1774	Resiniferatoxin	Capsaicin analog isolated from *Euphorbia*; PKC and TRPV agonist.
T6902	Tranilast	Mast cell stabilizer; TRPV2 antagonist, PDGFR inhibitor.
C0266	Capsaicin, natural	Found in *Capsicum*; TRPV agonist.
G3455	Ginsenoside Rc	Triterpene saponin found in species of *Panax*; AMPK inhibitor, potential TRPV1 antagonist.
G3456	Ginsenoside Rd	Triterpene saponin found in species of *Panax*; 26S proteasome inhibitor, TRPM7 antagonist.
A9818	Azelastine Hydrochloride	TRPV1 agonist, histamine H1 antagonist.
F4483	Flufenamic Acid	NSAID; TREK1 K^+ potentiator, voltage-gated Na^+ channel blocker, TRPC3 and TRPM2 antagonist, COX-1/2 inhibitor.
L5751	Lomerizine Dihydrochloride	L-type and T-type Ca^{2+} and TRP antagonist.
M1979	Methyl Salicylate	Ester found in *Spiraea, Betula*, and *Gaultheria*; TRPV1 modulator.
P7023	Pregnenolone	Endogenous precursor to all steroid hormones; TRPM3 agonist, NMDA potentiator, GABA-A negative allosteric modulator.
A7085	Arvanil	CB1 agonist, TRPV1 agonist.
E7556	Etodolac	NSAID; TRPA1 agonist, COX-2 inhibitor.
P3465	Piperine	Alkaloid found in black and long peppers; TRPV1 agonist.
S8151	Sumatriptan Succinate	Tryptamine; 5-HT1B/1D agonist, TRPV1 antagonist.
N5211	Nocistatin	Neuropeptide; TRPC agonist, BK/SK K^+ channel modulator.
P0270	Parthenolide	Sesquiterpene lactone found in *Tanacetum*; TRPA1 partial agonist, NLRP3 and caspase 1 inhibitor.
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^+ channel blocker, GLT-1 modulator.
K1678	Ketanserin	5-HT2A antagonist, potential α1-adrenergic antagonist.
P3348	Pimecrolimus	Calcineurin inhibitor, potential TRPV1 agonist.
N1755	Neomycin Sulfate	Polycationic aminoglycoside; protein translation inhibitor, cation channel/receptor (P2X, TRPV1) blocker.
P1634	Peimine	Steroidal alkaloid found in *Fritillaria*; TRPV1 and TRPA1 antagonist.
P1635	Peiminine	Steroidal alkaloid found in *Fritillaria*; M2 mAChR antagonist, potential TRPV1 and TRPA1 antagonist.
K1679	(+)-Ketanserin Tartrate Salt	5-HT2A and TRPV1 antagonist, potential α1-adrenergic antagonist.
A7200	AS-252424	p110γ PI3K inhibitor, potential TRPC1/5/6 negative modulator, potential TRPC3/7 positive modulator.
P3542	PIK-93	p110α and p110γ PI3K inhibitor.
G3252	6-Gingerol	Phenol found in *Zingiber*; TRPV1 agonist, 5-HT3 antagonist, TASK-1/TASK-3/TRESK K^+ channel blocker.
C4417	Clemizole	TRPC5 activator, NS4B and histamine H1 inhibitor.
C4418	Clemizole Hydrochloride	TRPC5, NS4B, histamine H1 inhibitor.
I0933	Icilin	TRPM8 activator, TRPV3 blocker.

NMDA

Product ID	Product Name	Description
A4443	L-(+)-Alliin	Optically active cysteine derivative found in *Allium*.
A5217	trans-Anethole	Phenylpropene derivative found in essential oils; NMDA agonist.
D1629	Dehydroepiandrosterone	Endogenous steroid hormone; ERβ, NMDA, σ1 agonist, ERα and AR partial agonist, GABA-A antagonist.
D1792	Dextromethorphan Hydrobromide Hydrate	σ1/2and μ/κ/δ-OR agonist, α3β4/α4β2/α7 nAChR, SERT, NET, NMDA antagonist.
S8247	Sulfasalazine	Mesalazine derivative, sulfa drug; sepiapterin reductase inhibitor, NMDA antagonist.
A4444	L-Alliin	Cysteine derivative found in *Allium*; NMDA NR2A/2B agonist.
D3328	Dihydrokainic Acid	NMDA agonist, GLT-1 inhibitor.
M1749	Memantine Hydrochloride	D2 agonist, NMDA, 5-HT3, α7 nAChR antagonist.
N3322	Niflumic Acid	NSAID; NMDA inverse agonist, T-type Ca^{2+} and Cl^- channel blocker, GABA-A antagonist, COX-1/2 inhibitor.
T2816	L-Theanine	Non-essential amino acid found in *Camellia* and *Boletus*, glutamic acid analog; weak AMPA and NMDA agonist.
A4802	Amantadine Hydrochloride	Viral M2 proton channel blocker, MAO-A, NET, NMDA, α7 nAChR antagonist.
A4803	Amantadine Sulfate	Viral M2 proton channel blocker, MAO-A, NET, NMDA, α7 nAChR antagonist.
P7023	Pregnenolone	Endogenous precursor to all steroid hormones; TRPM3 agonist, NMDA potentiator, GABA-A negative allosteric modulator.
C2951	Chlortetracycline Hydrochloride	Tetracycline; protein translation inhibitor, MMP and calpain inhibitor, NMDA antagonist.
C9610	D-Cycloserine	NMDA partial agonist, D-Ala-D-Ala ligase inhibitor.
H8162	(-)-Huperzine A	Sesquiterpene alkaloid found in *Huperzia serrata*; AChE inhibitor, NMDA antagonist.
A7656	Atomoxetine Hydrochloride	NET and SERT inhibitor, NMDA antagonist.
D1643	Delta Sleep Inducing Peptide	Peptide; GABA potentiator, NMDA negative allosteric modulator.
N1721	Nefiracetam	Pyrrolidone; NMDA and mGluR5 agonist, N-type and L-type Ca^{2+} channel activator, α4β2 nAChR potentiator.
C4558	Clonidine Hydrochloride	Imidazoline and α2-adrenergic agonist, Nav1.7 Na^+ channel blocker.
F4583	Flupirtine Maleate	Kv7 K^+ channel activator, NMDA antagonist, GABA-A negative modulator.
F4883	Flutriafol	Triazole; demethylation inhibitor, NMDA agonist.
G1855	Gentiopicroside	Secoiridoid glycoside found in *Gentiana* and *Cephalaria*.
I7559	Isoliquiritigenin, natural	Chalcone; SIRT activator, GABA-A positive modulator; NMDA antagonist, VEGFR2 and HDAC inhibitor.
R3197	Rhyncholphylline	Oxyindole alkaloid found in *Uncaria sinensis*; hERG K^+ and L-type Ca^{2+} channel channel blocker, NMDA antagonist.
S1853	Senegenin	Found in *Polygata tennifolia*.
P6870	Propofol	GABA-A agonist, NMDA antagonist, voltage-gated Na^+ channel blocker.
I7258	Isoflurane	Ca^{2+} ATPase and ATP-sensitive K^+ channel activator, GABA-A and GlyR potentiator, NMDA antagonist, L-type Ca^{2+}, delayed rectifier K^+, A-type K^+ channel blocker.
B3211	(+)-Bicuculline	NMDA potentiator, GABA-A antagonist.
R3347	Riluzole	Benzothiazole; TRPC5 agonist, PTR1 inhibitor, voltage-gated Na^+ channel blocker, GLT-1 modulator.
D3349	Dimebon Dihydrochloride	AMPK activator, L-type Ca^{2+} channel and NMDA, histamine H1/2, α-adrenergic, 5-HT2C/5A/6 receptor antagonist.
M1444	MDL 29951	GPR17 agonist, NMDA and fructose 1,6-bisphosphatase inhibitor.

AMPA

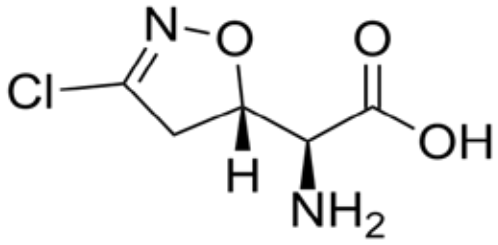
Product ID	Product Name	Description
K0133	Kainic Acid	Excitatory amino acid found in seaweed; AMPA and kainate agonist.
T2816	L-Theanine	Non-essential amino acid found in *Camellia* and *Boletus*, glutamic acid analog; weak AMPA and NMDA agonist.
A5326	Aniracetam	AMPA positive allosteric modulator, D2, 5-HT2A, nAChR agonist.
T7133	Trimetazidine	Long-chain 3-ketoacyl-CoA thiolase inhibitor, potential kainate and AMPA antagonist.
N5605	Nobiletin	Polymethoxylated flavone found in citrus fruits; potential AMPA positive modulator.
Z5852	Zonampanel	AMPA antagonist.
D3349	Dimebon Dihydrochloride	AMPK activator, L-type Ca^{2+} channel and NMDA, histamine H1/2, α-adrenergic, 5-HT2C/5A/6 receptor antagonist.
A5061	Ampalex	Benzylpiperidine; AMPA potentiator.

GABA

Product ID	Product Name	Description
D1629	Dehydroepiandrosterone	Endogenous steroid hormone; ERβ, NMDA, σ1 agonist, ERα and AR partial agonist, GABA-A antagonist.
T0081	Taurine	Endogenous sulfonic acid involved in Ca^{2+} signaling and cardiovascular function; GABA-A and GlyR agonist.
N3310	Nicotinamide	Amide form of vitamin B3 required for production of NAD and NADP; GABA modulator.
B3345	(-)-Bilobalide	Sesquiterpene lactone found in *Ginkgo*; GABA-A antagonist.
G3357	Ginkgolide C	Diterpene lactone found in *Ginkgo*; GABA-A, α-1 GlyR, PAF antagonist.
P8117	Puerarin, 99%	Isoflavone found in *Pueraria*; 5-HT2C and GABA-A antagonist.
B0110	Baclofen	GABA derivative; GABA-B agonist.
G0048	γ-Amino Butyric Acid	Endogenous neurotransmitter; GABA agonist.
Y0052	Yangonin	Dienolide kavalactone found in *Piper methysticum* (kava plant); CB1 agonist, GABA-A potentiator.
G3556	Ginsenoside Rg3	Triterpene saponin found in species of *Panax*; γ2 GABA-A agonist, Kv7.1 K^+ channel activator, α10 nAChR antagonist.
P6957	Protopanaxadiol	Triterpene sapogenin found in species of *Panax*; GABA-A antagonist.
P6958	Protopanaxatriol	Triterpene sapogenin found in species of *Panax*; GABA-A/C antagonist, slow-activating delayed rectifier K^+ channel blocker.
H5654	Honokiol	Lignan found in species of *Magnolia*; GABA-A potentiator.
M0125	Magnolol	Lignan found in *Magnolia*; GABA-A potentiator.
M1622	Mefenamic Acid	NSAID; GABA-A potentiator, COX-1/2 inhibitor.
N3322	Niflumic Acid	NSAID; NMDA inverse agonist, T-type Ca^{2+} and Cl^- channel blocker, GABA-A antagonist, COX-1/2 inhibitor.
G0106	Gabapentin	GABA analog; GABA potentiator, adenosine A1 agonist, voltage-gated α2δ Ca^{2+} channel blocker, NMDA modulator.
P8118	Puerarin	Isoflavone daidzein derivative found in *Pueraria*; GABA-A potentiator, 5-HT2C antagonist.
T2816	L-Theanine	Non-essential amino acid found in *Camellia* and *Boletus*, glutamic acid analog; weak AMPA and NMDA agonist.
C0270	Carbamazepine	GABA potentiator, voltage-gated Na^+ and ATP-sensitive K^+ channel blocker.
F4681	Flumazenil	GABA-A antagonist.
F8270	Furosemide	Loop diuretic; NKCC symporter inhibitor, CFTR Cl^- channel blocker, GABA-A antagonist.
I8618	Ivermectin	Avermectin; glu-gated Cl^- channel activator, GlyR and GABA-A agonist.
P7023	Pregnenolone	Endogenous precursor to all steroid hormones; TRPM3 agonist, NMDA potentiator, GABA-A negative allosteric modulator.
S1609	Securinine	Alkaloid found in *Securinega*; potential GABA-A antagonist.
A0501	Abamectin	Macrocyclic lactone avermectin; GABA antagonist, F0F1-ATPase and adenine nucleotide translocator inhibitor.
E4902	Emamectin B1 Benzoate	Semi-synthetic avermectin; GABA potentiator.
E6470	Eprinomectin	Semi-synthetic avermectin; GABA potentiator.
D1643	Delta Sleep Inducing Peptide	Peptide; GABA potentiator, NMDA negative allosteric modulator.
P1952	Penitrem A	Neuroactive mycotoxin produced by *Penicillum* and *Aspergillus*; GABA-A potentiator, BK K^+ channel inhibitor.
V1870	Verruculogen	Mycotoxin produced by *Aspergillus*; BK K^+ channel blocker, GABA-A antagonist.
V0147	Valproic Acid Sodium Salt	T-type Ca^{2+} and voltage-gated Na^+ channel blocker, GABA transaminase and HDAC inhibitor.
B8248	Bumetanide	Loop diuretic; NKCC symporter and KCC2 co-transporter inhibitor, potential GABA-A antagonist.
F4583	Flupirtine Maleate	Kv7 K^+ channel activator, NMDA antagonist, GABA-A negative modulator.
G3552	20S-Ginsenoside Rg3	Triterpene saponin found in *Panax*; Kv7.1 K^+ channel activator, γ2 GABA-A agonist, α10 nAChR antagonist.
F4881	Flumequine Sodium	Fluoroquinolone; bacterial DNA gyrase inhibitor, GABA-A antagonist.
B3211	(+)-Bicuculline	NMDA potentiator, GABA-A antagonist.
G4400	Glabridin	Isoflavonoid found in *Glycyrrhiza*; GABA-A positive modulator.
I7559	Isoliquiritigenin, natural	Chalcone; SIRT activator, GABA-A positive modulator; NMDA antagonist, VEGFR2 and HDAC inhibitor.
E7758	Etomidate	GABA-A agonist.
P6870	Propofol	GABA-A agonist, NMDA antagonist, voltage-gated Na^+ channel blocker.
I7258	Isoflurane	Ca^{2+} ATPase and ATP-sensitive K^+ channel activator, GABA-A and GlyR potentiator, NMDA antagonist, L-type Ca^{2+}, delayed rectifier K^+, A-type K^+ channel blocker.
R5874	Rosmarinic Acid	Caffeic acid ester isolated from *Melissa, Salvia*, and *Rosmarinus*; GABA transaminase and Fyn kinase inhibitor.
A8812	AWD 131-138	GABA-A positive modulator.
N7200	NS-11394	GABA-A α5/3/2 positive modulator.

Acivicin

Acivicin (A0934) is a heterocyclic analog of L-glutamate originally produced by *Streptomyces sviceus*. Acivicin exhibits antimicrobial and anticancer activities.



A0934 Acivicin

Acivicin inhibits γ-glutamyltranspeptidase (γ-GT), an enzyme involved in transferring γ-glutamyl groups in the cell membranes of the kidneys, heart, brain, and pancreas. γ-GT moves amino acids across cell membranes and is important for homeostasis of oxidative stress. Acivicin covalently binds the active site of γ-GT, inducing a conformational change in which the C-terminal end folds to block entry and exit to the active site; this renders the enzyme incapable of catalyzing other reactions[1]. As glutathione is hydrolyzed by γ-GT, acivicin is often used to study glutathione homeostasis.

Acivicin inhibits glutamine amidotransferases such as GMP synthase and IGP synthase. Acivicin suppresses the growth of hepatoma cells in vitro, preventing the incorporation of uridine and thymidine into macromolecules and decreasing pools of CTP, GTP, dCTP, dGTP, and tTTP[2]. In this study, acivicin also inhibits CTP synthase. Other studies indicate depletion of all nucleoside triphosphate pools[3].

Acivicin also exhibits chemotherapeutic activity in clinical trials, although potential benefit has been limited by CNS toxicity[4]. This compound has also been studied as a low-dose supplement to other chemotherapeutics such as cisplatin, as inhibition of γ-GT suppresses cisplatin-induced nephrotoxicity[5].

Acivicin's inhibition of GMP synthase also limits its pathogenicity of fungal microbes *Candida albicans* and *Aspergillus fumigatus*[6]. Analogs of acivicin are in various stages of development as antiparasitic compounds, owing to their increased affinity for CTP synthase[7].

References:
1. Williams K, Cullato S, Sand A, et al. Biochemistry. 2009 Mar 24;48(11):2459-67.
2. Lui MS, Kizaki H, Weber G. Biochem Pharmacol. 1982 Nov 1;31(21):3469-7
3. Weber G, Natsumeda Y, Lui MS, et al. Adv Enzyme Regul. 1984;22:69-93.
4. Hidalgo M, Rodriguez G, Kuhn JG, et al. Clin Cancer Res. 1998 Nov;4(11):2763-70.
5. Hanigan MH, Gallagher BC, Taylor PT Jr. Am J Obstet Gynecol. 1996 Aug;175(2):270-4.
6. Rodriguez-Suarez R, Xu D, Veillette K, et al. Chem Biol. 2007 Oct;14(10):1163-75.
7. Tamborini L, Pinto A, Smith TK, et al. ChemMedChem. 2012 Sep;7(9):1623-34.

ALK Inhibitors

Tyrosine kinases such as anaplastic lymphoma kinase (ALK) are becoming major targets in the development of new chemotherapeutics and anti-inflammatories. ALK plays an important role in the development of the brain; it also drives the progression of several cancers, including anaplastic large-cell lymphoma, neuroblastoma, and non-small cell lung cancer.

When the ALK gene is mutated or fused with other genes, it often produces extra or abberent proteins. Overactive ALK stimulates JAK/STAT, PI3K/Akt, and ERK, promoting unregulated cell cycle progression, survival, and proliferation[1]. Targeting ALK prevents these downstream effects, limiting cancer cell signaling and tumor growth.

Several ALK inhibitors also inhibit IGF-1R, an additional target in preventing growth of cancer cells[2-4]. Products that target both of these kinases include **LDK378 (L1340)**, **AZD3463 (A9600)**, and **GSK-1838705A (G7540)**.

References:
1. Grande E, Bolós MV, Arriola E. Mol Cancer Ther. 2011 Apr;10(4):569-79.
2. www.clinicaltrials.gov/show/NCT01685060
3. Yang B. Protein Kinases in Drug Discovery Conference. 2013.
4. Sabbatini P, Korenchuk S, Rowand JL, et al. Mol Cancer Ther. 2009 Oct;8(10):2811-20.
5. Okamoto W, Okamoto I, Arao T, et al. Mol Cancer Ther. 2012 Jul;11(7):1557-64.
6. Kodama T, Hasegawa M, Takanashi K, et al. Cancer Chemother Pharmacol. 2014 Nov;74(5):1023-8.
7. Hoogendijk AJ, Pinhanços SS, van der Poll T, et al. Immunobiology. 2013 Apr;218(4):435-42.
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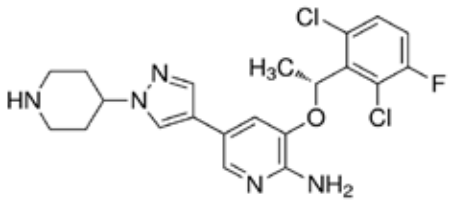

Crizotinib (C6935) is a well-characterized ALK inhibitor that also suppresses activity of ROS1 and c-MET. In cancer cells, this compound upregulates expression of pro-apoptotic BIM and downregulates expression of anti-apoptotic survivin to induce apoptosis[5].

CH5424802 (C2900) targets both wild-type and mutant L1196M ALK, inducing regression of non-small cell lung cancer metastasis in the brain[6].

Doramapimod (D5868) is an inhibitor of ALK, JNK, and p38 MAPK that suppresses pulmonary inflammation in vivo and in vitro[7].

ASP-3026 (A7400) is an inhibitor of ALK that lowers tumor burden in lung and intrapleural tumor models[8].

Allicin

Allicin (A4440) is an organosulfur compound found in garlic. Like other organosulfurs, allicin exhibits a wide variety of biological activities, including antiviral, antibacterial, anti-inflammatory, antioxidative, antihypertensive, and anticancer properties.

LKT Laboratories now carries an aqueous solution of allicin (**Allicin, aqueous A4441**) that eliminates the presence of methanol as a solvent. This solution of water and 0.1% formic acid allows allicin to be used in cell cultures and animal studies without the cytotoxic effects of methanol. The aqueous solution does not sacrifice quality or stability for its ease-of-use benefit. General storage conditions and stabilities are similar to those of allicin in methanol.

Several studies using allicin supplied from LKT Laboratories have been published within the last several years, many focusing on the antimicrobial effects of allicin. One study highlights the ability of allicin to inhibit streptolysin O, a potent cytolytic toxin produced by species of *Streptococcus*, a gram-positive bacteria[1].

References:
1. Arzanlou M, Bohlooli S. J Med Microbiol. 2010 Sep;59(Pt 9):1044-9.
2. Lihua L, Jianhuit W, Jialini Y, et al. Pol J Microbiol. 2013;62(3):243-51.
3. Hasan N, Siddiqui MU, Toossi Z, et al. Biochem Biophys Res Commun. 2007 Apr 6;355(2):471-6.
4. Liu C, Cao F, Tang QZ, et al. J Nutr Biochem. 2010 Dec;21(12):1238-50.
5. Reinhart KM, Coleman CI, Teevan C, et al. Ann Pharmacother. 2008 Dec;42(12):1766-71.
6. Chu YL, Ho CT, Chung JG, et al. J Agric Food Chem. 2012 Aug 29;60(34):8363-71.
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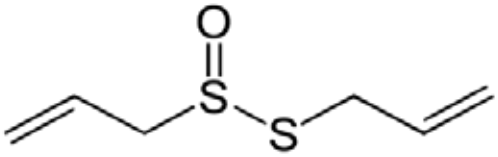


Allicin, aqueous (A4441) does not contain methanol, making it easier to use in cell culture and animal studies!

A separate study suggests that allicin inhibits surface adherence and suppresses production of virulence fctors by gram-negative bacteria *Pseudomonas aeruginosa*[2]. Additionally, allicin downregulates expression and secretion of *Mycobacterium tuberculosis* 85B mRNA in infected monocytes[3]. This effect is likely due to allicin's anti-inflammatory and antioxidative activities, as it increases levels of glutathione and IFN-γ and decreases levels of TNF-α.

Allicin displays other antioxidative activities as well. In animal models, allicin suppresses increased ROS levels and NADPH activity induced by angiotensin II or pressure overload, preserving cardiac function and preventing the development of cardiac hypertrophy[4]. This compound also reduces systolic and diastolic blood pressure when administered to hypertensive subjects in a clinical setting[5].

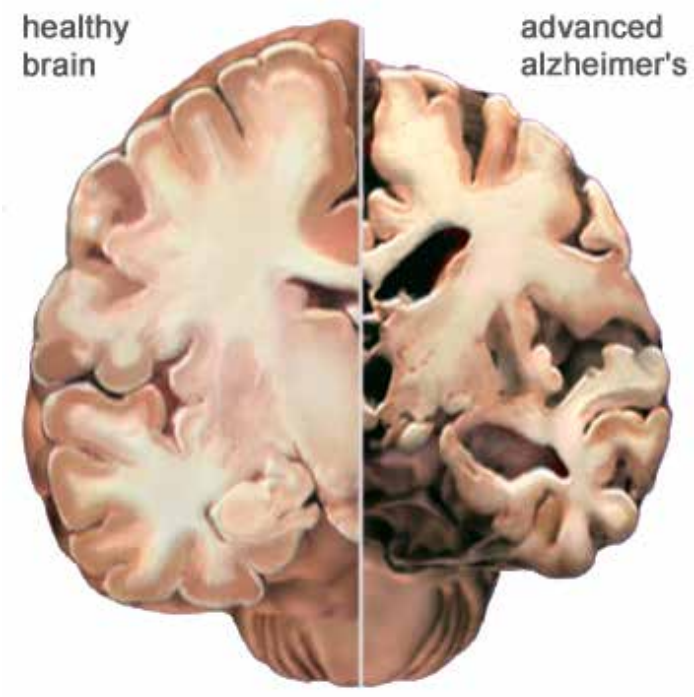
In various cellular and animal models, allicin also exhibits anticancer and chemopreventive properties. This compound suppresses PI3K/mTOR signaling and induces p53-mediated autophagy in hepatocellular carcinoma cells[6]. Allicin also inhibits growth of glioma cells in vitro through modulation of MAPK/ERK signaling and Bcl-2/Bax levels[7].

Linking Alzheimer's Disease and Insulin Signaling

Research has indicated a potential link between Alzheimer's disease (AD) and insulin resistance for several years. Commonalities between AD and type 2 diabetes mellitus (T2DM) are highlighted by interruptions in insulin signaling that affect cell growth, cell differentiation, cellular repair mechanisms, energy metabolism, and glucose utilization[1]. Insulin is primarily known for regulating blood sugar levels but also plays a significant role in neuronal function as it acts as a growth factor for both regular cells as well as neurons.

Recent research highlights links between AD and increased resistance to insulin in the brain. Insulin resistance appears to be an early, common feature of AD and is accompanied by insulin-like growth factor (IGF) resistance[2]. This resistance develops in the brains of subjects with AD even without the presence of T2DM.

By restoring insulin responsiveness and lowering insulin depletion in the brain, studies suggest that it may be possible to slow or halt the effects of AD. Pharmacological agents that can perform these functions are currently under investigation and are being studied for their effects in T2DM models. **Metformin Hydrochloride (M2076)**is a commonly used treatment for T2DM.



Metformin is an insulin sensitizer that decreases hepatic glucose production. In pre-clinical studies, metformin sensitizes neuronal insulin resistance[3].

LKT Laboratories carries a variety of antidiabetic compounds. Other potentially useful antidiabetic therapies include PPAR agonists **Pioglitazone Hydrochloride (P6954)** and **Troglitazone (T7056)** and ATP-sensitive K^+ channel modulators **Glimepiride (G4535)** and **Repaglinide (R1860)**.

References:
1. Gao C, Liu Y, Li L, et al. Rev Neurosci. 2013;24(6):607-15.
2. Talbot K, Wang HY, Kazi H, et al. J Clin Invest. 2012 Apr;122(4):1316-38.
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Antipsychotics and Antidepressants

LKT Laboratories offers a variety of bioactive antipsychotic and antidepressant compounds for use in research models. Many of these compounds alter dopamine signaling, inhibiting one of several dopamine receptor subtypes or dopamine transporters. LKT Laboratories carries typical first generation antipsychotics such as **Chlorpromazine Hydrochloride (C2947)** and **Haloperidol (H0142)** as well as atypical second generation antipsychotics **Clozapine (C4757)** and **Risperidone (R3475)**.

A second category of neuroactive compounds includes antidepressants, which typically act to prolong the activity of neurotransmitters in the brain. These compounds often act on serotonin receptors, dopamine receptors, ion channels, and neurotransmitter transporters. Selective serotonin reuptake inhibitors (SSRIs) inhibit presynaptic reuptake of serotonin from the synapse back into the neuron, increasing levels of serotonin available to interact with serotonin receptors. **Fluoxetine Hydrochloride (F4780)** and **Paroxetine Hydrochloride Hemihydrate (P0297)** are SSRIs used in many research applications.

Monoamine oxidase inhibitors (MAOIs) are another category of antidepressants that inhibit monoamine oxidase, an enzyme responsible for degradation of serotonin and dopamine. Inhibition of this enzyme increases levels of these neurotransmitters, allowing them to bind receptors and transmit signals involved in reward, reinforcement, learning, and memory. **Tranylcypromine Hydrochloride (T6903)** and **Moclobemide (M5610)** are two of several MAOIs carried by LKT Laboratories.

Additional representative antipsychotics and antidepressants from our product library can be found in the listing below.

A5234 Amisulpride
A5235 Amitriptyline Hydrochloride
A5059 Amoxapine
A7034 Aripiprazole
A9801 Azaperone
B9363 Bupropion Hydrochloride
B8274 Buspirone Hydrochloride
C3477 Citalopram Hydrobromide
C4457 Clomipramine Hydrochloride
D1874 Desvenlafaxine Succinate
D5994 Doxepin Hydrochloride
D8145 Duloxetine Hydrochloride
E7209 Escitalopram Oxalate
F4584 Fluphenazine
F4783 Fluvoxamine Maleate
I4659 Iloperidone
K1678 Ketanserin
M0262 Maprotiline Hydrochloride
M1845 Melitracen Hydrochloride
M3368 Mirtazapine
N1822 Nefazodone Hydrochloride
O4400 Olanzapine
P0144 Paliperidone
Q8019 Quetiapine Fumarate
S1971 Sertraline Hydrochloride
T2936 Thioridazine Hydrochloride
T7003 Trazodone Hydrochloride
T7033 Trifluoperazine Hydrochloride
V1854 Venlafaxine Hydrochloride
V3444 Vilazodone
V5870 Vortioxetine
Z3463 Ziprasidone

Proteins Involved in Apoptosis

Apoptosis is a form of programmed cell death in which the cell signals through various pathways for its own destruction. Apoptosis is a normal process involved in development but can also be initiated in response to stressors such as hypoxia, ionic imbalance, and pathogenic infection. Dysregulation of apoptosis plays a role in various diseases such as atrophy, neurodegeneration, autoimmune diseases, and cancer. In cancer, apoptosis is often inhibited, allowing a cell to live longer than planned. This allows the cell to pass on age-related misfolded proteins as it continues to replicate. In cancerous cells, apoptosis-regulating proteins are often mutated or dysregulated, especially cell cycle regulators such as p53 or apoptosis inhibitors such as Bcl-2 and XIAP. p53 is a tumor suppressor that inhibits cell cycle progression at the G1 phase in response to DNA damage; once halted, p53 either signals for DNA repair or apoptosis. Mouse double minute 2 homolog (MDM2) and other MDM-family proteins are negative regulators of p53; inhibiting these proteins allows p53 activity. Bcl-family proteins such as Bcl-2 and Bcl-xl inhibit apoptosis by preventing cytochrome C release and caspase activation, two steps in the initiation of apoptosis signaling cascades. Inhibitor of apoptosis proteins (IAPs) such as XIAP and survivin are also pro-survival; these proteins directly bind caspases, preventing caspase activation.

Bcl Inhibitors

Product ID	Product Name	Description
G5875	Gossypol Acetic Acid	Bcl-2, Bcl-xl, sialyl transferase inhibitor.
A0776	ABT-199	BH3 mimetic; Bcl-2 inhibitor.
A0777	ABT-263 (Navitoclax)	BH3 mimetic; Bcl-2 and Bcl-xl inhibitor.
A0778	ABT-737	BH3 mimetic; Bcl-2, Bxl-xl, Bcl-w inhibitor.
O0400	Obatoclax	BH3 mimetic; Bcl-2 and Bcl-xl inhibitor.

IAP Inhibitors

Product ID	Product Name	Description
L1044	LCL-161	SMAC mimetic; PXR agonist, IAP inhibitor.
T4400	TL-32711	Smac mimetic; IAP inhibitor.
Y4800	YM-155	Survivin inhibitor.

Other Apoptosis Activators

Product ID	Product Name	Description
D3450	Dimethylaminoparthenolide	Water-soluble parthenolide analog; NF-κB inhibitor.
J5237	JNJ-26854165	MDM2 inhibitor.
N7209	NSC-207895	Benzofuroxan derivative; MDMX inhibitor.
N8277	Nutlin-3	Imidzoline derivative; MDM2 inhibitor.
R3476	RITA	p53 activator.

Aurora Kinase Inhibitors

Aurora kinases are a class of enzymes responsible for regulating cell cycle progression. During mitosis, these proteins help facilitate chromosome segregation and cytokinesis. Aurora kinases are overexpressed in cancer cells, stimulating tumor progression; as a result, this group of proteins makes an excellent target in the development of new chemotherapeutics.

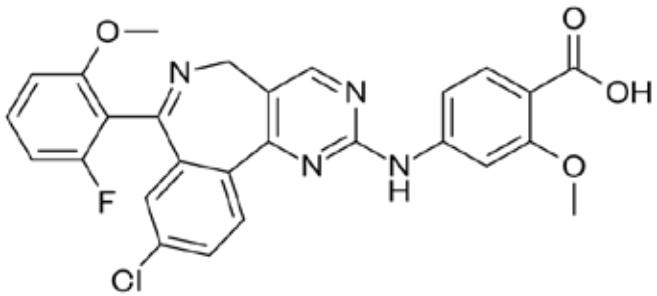
Aurora kinase A, one of three subtypes along with aurora kinases B and C, plays a signficant role in p53 signaling. The p53 signaling pathway is involved in cell growth regulation and apoptosis. The p53 protein functions as a tumor suppressor. Overexpression of aurora kinase A stimulates degradation of p53, downregulating checkpoint response signaling and facilitating oncogenic transformation in cells[1].

Aurora kinase B inhibition induces catastrophic mitosis. By preventing kinetochores from fixing inappropriate microtubule interactions, inhibition of this kinase prevents chromosome alignment and segregation, blocking cell division[2]. Aurora kinase inhibitors do not directly inhibit cell cycle progression but instead, following an aberrant mitosis, activate the p53-dependent post-mitotic checkpoint; this induces 'pseudo-G1' cell cycle arrest[3]. Because of this, highly abnormal cells continue proliferation despite the presence of significant genomic instabilities, resulting in cell death.

LKT Laboratories carries a variety of aurora kinase inhibitors. See the list below for a selection of these compounds.

Aurora kinase inhibitors:
A9714 AZD-1152-HQPA
C9708 CYC-116
G7444 GSK-1070916
M4652 MLN8237 (Alisertib)
T5996 Tozasertib
V9201 VX-11e
Z4900 ZM-447439

References:
1. Katayama H, Sasai K, Kawai H, et al. Nat Genet. 2004 Jan;36(1):55-62.
2. Ditchfield C, Johnson VL, Tighe A, et al. J Cell Biol. 2003 Apr 28;161(2):267-80.
3. Harrington EA, Bebbington D, Moore J, et al. Nat Med. 2004 Mar;10(3):262-7.



M4652 MLN8237 (Alisertib)

Autophagy Inducers

Autophagy is the breakdown of unnecessary or dysfunctional cell components by lysosomes. This process can promote survival during cellular starvation by maintaining cellular energy levels. Autophagy is also a form of programmed cell death that can be initiated by processes such as development, differentiation, neurodegenerative diseases, stress, infection, and cancer[1]. Although autophagy may improve survival of starved cells, it is also a tumor suppressor. Autophagy is induced by the presence of irregular proteins produced by chemotherapeutics; degradation of these proteins and organelles limits cell growth[2]. Prolonged autophagy also leads to a high turnover rate of proteins and organelles which may kill cancer cells[3]. For a selection of compounds that induce autophagy across various disease models including cancer, fibrosis, and pathogenic infection, see the representative list below.

A0820 Acetyl-benzylisothiocyanate-L-cysteine
A4440 Allicin
A4441 Allicin (aqueous)
A6132 Apicidin
A6818 Arenobufagin
A7460 Asparaginase
A9710 AZD-2014
B1653 Benzyl Isothiocyanate
B1996 BEZ235
C0265 Carnosic Acid
C2947 Chlorpromazine Hydrochloride
C5654 Concanavalin A
D0375 Dasatinib Monohydrate
D4802 17-DMAG
E0813 Ecdysterone
E2003 Efavirenz
E6846 Erlotinib Monohydrochloride
E7357 Esomeprazole Potassium
E7356 Esomeprazole Magnesium Trihydrate
E7657 Etoposide
E7658 Etoposide phosphate
E8419 Everolimus
G3461 Ginsenoside F2
H9712 (E)-4-Hydroxytamoxifen
H9711 (Z)-4-Hydroxytamoxifen
H9716 (E,Z)-4-Hydroxytamoxifen
M1744 Melittin
M4454 MLN-4924
N3346 Nilotinib
O0400 Obatoclax
O6932 Oridonin
P3465 Piperine
P3561 Piperlongumine
P4492 PLX4720
P7000 PR-619
P7219 Pseudolaric acid B
P8167 Puromycin Aminonucleoside
P8168 Puromycin Dihydrochloride
P9200 PX-866
S0134 Saikosaponin D
S7603 Stavudine
S8044 R,S-Sulforaphane
T1968 Terpinen-4-ol
V0146 Valsartan
V0352 Vandetanib
W5727Wogonoside
Y4802 YM-201636

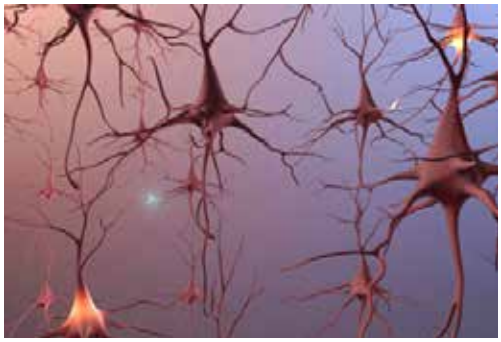
References:
1. Kobayashi S. Biol Pharm Bull. 2015;38(8):1098-103.
2. Mathew R, Karp CM, Beaudoin B, et al. Cell. 2009 Jun 12;137(6):1062-75.
3. Yang ZJ, Chee CE, Huang S, et al. Mol Cancer Ther. 2011 Sep;10(9):1533-41.

Bexarotene

Bexarotene (B1992) is a retinoid specifically selective for retinoid X receptors (RXRs). Bexarotene is a chemotherapeutic agent in the treatment of cutaneous T cell lymphoma. Research also suggests that bexarotene exhibits potential biological activity in the treatment of Alzheimer's disease as well.

The correlation between amyloid-β plaques and Alzheimer's disease is well established[1]. The accumulation and aggregation of misfolded plaques interferes with neuronal function and neurotransmission by inhibiting the transfer of signals in the brain. Amyloid-β peptides are produced by cleavage of amyloid precursor protein (APP), a transmembrane glycoprotein. Although the normal functions of amyloid-β are not well understood, it is primarily aggregation of the misfolded fragments that is linked to the development of Alzheimer's disease.

Apolipoprotein E is a protein that is responsible for facilitating the clearance of amyloid-β plaques. Transcriptional activation of apolipoprotein E is normally induced through activity at nuclear receptors such as RXRs. Bexarotene, as an RXR agonist, enhances the clearance of soluble amyloid-β within hours of administration in animal models of Alzheimer's disease[2]. Amyloid-β plaque area is reduced by more than 50% within three days. Bexarotene also stimulates the reversal of cognitive deficits and improves neural ciruit function.



Healthy neurons

Neurons surrounded by amyloid-β plaques

Further research with bexarotene examining its activity in Alzheimer's disease models and other research applications is ongoing. As RXR activation stimulates physiological amyloid-β clearance mechanisms, bexarotene and other RAR agonists such as **9-cis-Retinoic Acid (R1777)** and **13-cis-Retinoic Acid (R1779)** may gain new relevance in neurodegenerative disease studies.

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Bleomycin

Bleomycins are glycopeptides first produced by *Streptomyces verticillus* that exhibit potent anticancer and antibiotic properties. **Bleomycin Sulfate (B4518),** a mixture of bleomycins, contains the predominant components of commercially available bleomycin. Bleomycin is used to treat many cancers, including squamous cell carcinoma, testicular cancer, and Hodgkin lymphoma; it is also a component of the ABVD chemotherapy regimen. Bleomycin induces strand breaks in DNA and prevents incorporation of thymidine into DNA[1-3].

Bleomycin has two primary structural domains: the bithiazole DNA interaction site and a metal binding site[1]. Bleomycin can chelate iron at the second site, generating reactive oxygen species that cause DNA degradation[4]. Bleomycin may also bind to DNA directly, inducing strand breakage by removing hydrogen from DNA bases[5].

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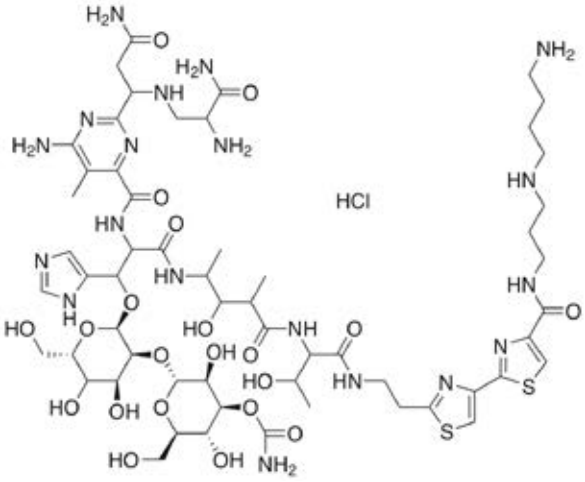

Bleomycin A5 HCl (A4517) can induce two modes of cell death: necrosis and apoptosis. Bleomycin A5 may also treat hemangioma, as it has shown activity in cellular and animal models[6]. Additionally, this compound upregulates expression of p53 and downregulates activity of telomerase in vitro[7].

Carotenoids

Carotenoids are compounds that can be found in photosynthetic organisms and are often used as pigments or dyes. Most humans and animals are incapable of synthesizing carotenoids and must obtain them through a diet of fruits and vegetables. Structurally, carotenoid compounds consist of a series of terpene units that allow for effective free radical scavenging. As antioxidants, carotenoids exhibit a variety of biological activities and health benefits, including risk reduction for breast cancer, prostate cancer, and gastric cancer.

β-Carotene (C0269) is a red-orange pigment found in sweet potatoes, carrots, pumpkins, and spinach; it exhibits provitamin A activity and can be cleaved into two molecules of vitamin A. β-Carotene intake is associated with reduced risk for amyotrophic lateral sclerosis (ALS), likely due to its ability to reduce oxidative stress, a component involved in the pathogenesis of ALS[1]. High dietary levels of β-carotene are also associated with reduced risk of breast cancer and gastric cancer[2-3]. Administration of β-carotene suppresses the invasive activity of hepatoma cells[4]. In animal models of mammary carcinogenesis, β-carotene intake in a lipid-enriched diet decreases tumor incidence and tumor growth[5].

Lycopene (L9609) is another red pigment with biological activity. Lycopene is found in many red fruits and vegetables such as tomatoes, melon, and grapefruit. Like other carotenoids, this compound is also an antioxidant. Intake of lycopene is associated with protection against UV-induced skin damage; subjects administered lycopene present increases in procollagen I and decreases in MMP-1, two biomarkers of UV-induced erythma[6]. In animal models of liver carcinogenesis, this compound minimizes diethylnitrosoamine-induced decreases in hepatic antioxidative enzyme activity, increases in inflammatory signaling, and activation of mTOR and NF-κB; as a result, the incidence, number, size, and volume of hepatic nodules is decreased[7].

Other carotenoids such as **Capsanthin (C0260)** exhibit additional research applications. This compound decreases expression of IL-6, TNF-α, and MCP-1, suppressing obesity-induced inflammation in adipocytes[8]. This compound also inhibits cell cycle progression and induces apoptosis in leukemia cells, potentially through the upregulation of PPARγ[9].

References:
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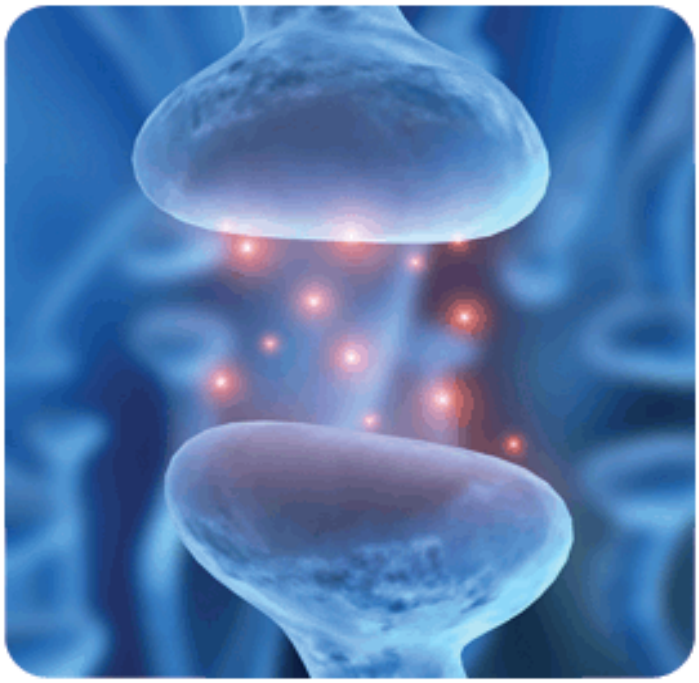
Cholinergic Signaling Modulators

Acetylcholine (ACh) is a neurotransmitter in both the peripheral nervous system (PNS) and central nervous system (CNS) in many organisms. ACh is one of many neurotransmitters in the autonomic nervous system and the only neurotransmitter found in the motor division of the somatic nervous system. ACh is also the principal neurotransmitter in autonomic ganglia. This compound activates signaling cascades at the neuromuscular junction and exerts effects on plasticity, arousal, reward, sensory perception, and attention[1-2].

LKT Laboratories offers several compounds that modulate ACh activity. **Darifenacin (D0169)** inhibits M3 muscarinic acetylcholines receptors[3]. **Donepezil Hydrochloride (D5753)** is a reversible acetylcholinesterase inhibitor that prolongs ACh activity and is used to treat Parkinson's disease[4]. **Pancuronium Bromide (P0252)** and **Atracurium Besylate(A7668)** are nonselective antagonists at nicotinic acetylcholine receptors at neuromuscular junctions and are used to induce muscle relaxation and anesthesia[5]. **Bupropion Hydrochloride (B8363)** is an antidepressant and smoking cessation aid that acts as a noncompetitive antagonist at $\alpha_3\beta_2$ and $\alpha_4\beta_2$ nicotinic acetylcholine receptors[6].



Mecamylamine Hydrochloride (M1708) is also an antagonist at nicotinic acetylcholine receptors, although it inhibits a much wider variety of receptor subtypes; it is primarily used in reward and reinforcement research models[7]. **Rivastigmine Hydrogen Tartrate (R3586)** is an inhibitor of acetylcholinesterase and butylcholinesterase that is used to treat dementia and to study Alzheimer's disease in research models[8].

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9-cis-Retinoic Acid

9-cis Retinoic Acid (R1777) is a form of vitamin A. 9-cis Retinoic acid is an endogenous ligand for retinoid X receptors, and like other retinoids, also activates retinoic acid receptors[1]. Retinoids such as 9-cis retinoic acid play a significant role in cell proliferation, cell differentiation, immune function, growth of bone tissue, and activation of tumor suppressor genes. This compound is one of the first commercially utilized retinoids; it displays therapeutic activity in the treatment of Kaposi's sarcoma and chronic hand eczema[2-3]. 9-cis Retinoic acid also exhibits potential benefit in the treatment of other cutaneous disorders such as pityriasis rubra pilaris[4].

9-cis Retinoic acid displays anticancer activity in the treatment of other cancers as well, including hormone-dependent tumors. Adrenocortical cancer is associated with reductions in endogenous retinoic acid production. In models of adrenocortical cancer, 9-cis retinoic acid decreases cell viability and steroid hormone secretion likely through disruption of cell cycle regulation. In animal models of this cancer, this compound decreases tumor growth[5]. In models of breast cancer, 9-cis retinoic acid suppresses cell proliferation by altering the interaction between retinoid X receptor α (RXRα) and replication factor C3 (RFC3); this appears to be the same mechanism by which this compound alters developmental embryonic cell proliferation[6].

9-cis Retinoic acid also exhibits antioxidative and neuroprotective activities. In models of neurotoxicity induced by methamphetamine administration, 9-cis retinoic acid improves locomotor activity and striatal tyrosine hydroxylase levels, limiting dopaminergic neurodegeneration[7]. This compound improves motor activity and minimizes neurodegenerative symptoms in animal models of Parkinson's disease as well[8].

This compound is relevant to many other additional research applications. Retinoids are known to play a key role in maintenance of immune function. In several cellular models, 9-cis retinoic acid stimulates immune cell adhesion in both integrin-dependent and integrin-independent manners[9]. Additionally, 9-cis retinoic acid enhances lymphatic vessel proliferation and regeneration in animal models, decreasing symptoms of lymphedema[10].

<u>Other retinoids available include:</u>
All-trans Retinol (R1876)
All-trans Retinol (high purity, R1877)
Trans-retinoic Acid (R1870)
13-cis Retinoic Acid (R1779)
Retinyl Palmitate (R1879)
Retinyl Acetate (R1878)
Etrtinate (E7668)
Acitretin (A0933)
And many others!

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Coffee Compounds

Cafestol (C0020) and **Kahweol (K0030)** are natural diterpenes found in coffee beans[1]. These compounds and their derivatives exhibit a variety of biological activities, including chemopreventive, anti-angiogenic, antioxidative, and anti-cancer properties.

Clinically, consumption of unfiltered coffee beverages is associated with a reduction in colon cancer prevalence[2]. In animal models of colon cancer, administration of **Cafestol Palmitate (C0021)** and **Kahweol Palmitate (K0032)** prevents the formation of DNA adducts by carcinogen PhIP; this may be a result of the ability of these compounds to increase expression of glutathione-S-transferase, a phase II detoxifying enzyme[3].

Cafestol and kahweol also exhibit anti-angiogenic and anti-inflammatory properties in many angiogenesis models. These compounds suppress tube formation and inhibit migration, invasion, and proliferation in endothelial cells[4-5]. Kahweol also inhibits expression of pro-inflammatory mediators COX-2 and MCP-1 in cellular models[5].

In other cellular models, kahweol inhibits differentiation of bone marrow-derived macrophages and monocytes into osteoclasts[6]. The inhibition of osteoclast development prevents bone resorption, improving bone strength and structure.

Also available:

C0021 Cafestol Acetate
C0025 Cafestol Eicosanate
C0027 Cafestol Linoleate
C0029 Cafestol Oleate
C0021 Cafestol Palmitate
C0033 Cafestol Stearate

K0031 Kahweol Acetate
K0034 Kahweol Eicosanate
K0036 Kahweol Linoleate
K0038 Kahweol Oleate
K0032 Kahweol Palmitate
K0040 Kahweol Stearate
And many others!

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Curcumin

Curcumin (C8069) is found in turmeric, a plant of the ginger family. Turmeric has traditionally been used to dye clothing and to preserve food. Along with curcumin, the two other primary curcuminoids present in turmeric are **Demethoxycurcumin (D1850)** and **Bisdemethoxycurcumin (B3573)**. These three compounds exhibit antioxidative, anticancer, anti-inflammatory, and antimicrobial properties.

Curcumin displays antiviral activity, inhibiting HIV-1 integrase activity in vitro[1]. HIV integrase is a key regulator of the retroviral life cycle. Additionally, curcumin inhibits HIV-1 replication[2].

Curcumin also exhibits neuroprotective activity. This compound prevents formation of soluble oligomers in amyloid-β-infused animal models[3]. Aggregation of amyloid-β (Aβ) fibrils into plaques is a key process in the progression of Alzheimer's disease. Curcumin accumulates near amyloid deposits in vivo; it also prevents the formation of new Aβ plaques and clears existing aggregations[4].

In cellular models of breast cancer, curcumin decreases metastatic activity. In this study, curcumin prevents transcription of CXCL1 and CXCL2, silencing activity of CXCR4 and other metastasis-promoting proteins[5].

Other studies highlight a different role for curcumin in cancer models. In one study, curcumin promotes the progression of lung lesions from benign hyperplasias to adenomas and carcinomas.

Available curcuminoids:
C8069 Curcumin
C8070 Curcumin (high purity)
D1850 Demethoxycurcumin
B3573 Bisdemethoxycurcumin
D3449 Dimethoxycurcumin
D3420 3,4-Difluorobenzocurcumin

When compared to BHT, a known carcinogen, administration of curcumin induces a similar increase in tumor growth and progression. This carcinogenic potential may be linked to modulation of ROS levels and oxidative stress in lung tissue[6]. This compound also disrupts the natural conformation of tumor suppressor p53, inhibiting its ability to bind DNA and induce cell cycle arrest[7].

In addition to curcumin, LKT Laboratories carries several other curcuminoids, including **3,4-Difluoro-benzocurcumin (D3420)**. This compound is in early stages of research but shows potential activity in a variety of biological applications as it exhibits better pharmacokinetic bioavailability and stronger pharmacological activity than curcumin[8].

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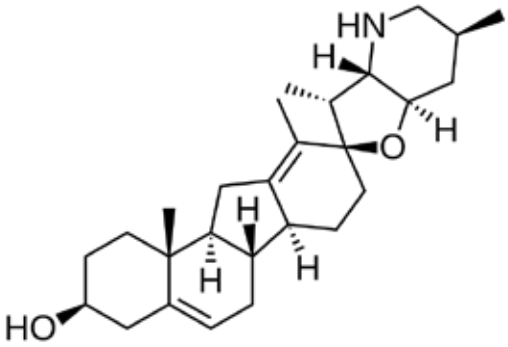
Cyclopamine

Cyclopamine (C9710) is a steroidal alkaloid isolated from the corn lily (*Veratrum californicum*) known for its inhibitory action on Hedgehog (Hh) signaling pathways. Across a variety of studies, cyclopamine exhibits several biological activities, including anticancer and anti-inflammatory properties.

The Hh signaling pathway regulates cell growth and differentiation, playing a significant role in embryonic development and tissue regeneration. Hh proteins bind and inhibit the Patched receptor, allowing levels of neighboring transmembrane receptor Smoothened (Smo) to increase and suppress cleavage (inactivation) of downstream transcription factors; the end result of this signaling cascade is gene activation. Signaling initiated by Hh proteins appears to be overactive in several types of cancer, potentially allowing unchecked cell growth[1-3].

Inhibition of proteins in the Hh signaling pathway shows benefit in the treatment of cancer in preclinical models. Mutations in Patched or Smo may result in constitutively active signaling. Gain-of-function mutations allow Smo to signal for gene transcription without input from upstream proteins and receptors; these mutations have been linked to the development of basal cell carcinoma and colorectal cancer[4-5].



C9710 Cyclopamine

Cyclopamine directly inhibits Smo, preventing Hh binding and the induction of gene transcription[6]. In one study, cyclopamine induces apoptosis in colorectal adenoma cells and colorectal carcinoma cells[7]. In a cellular model of cholangiocarcinoma, cyclopamine inhibits the epithelial-to-mesenchymal transition as well as cell migration, invasion, and proliferation[8]. Similarly, cyclopamine administered to animal models increases cell necrosis and inhibits tumor growth.

Cyclopamine also displays other biological activities. In an animal model of cholestasis due to hepatic ischemia/reperfusion injury, administration of cyclopamine decreases Akt and ERK activation, reduces neutrophil infiltration, and lowers levels of pro-inflammatory cytokines and fibrosis biomarkers; it also reduces overall histological damage[9]. In separate models of kidney fibrosis, this compound decreases levels of fibronectin and collagen I, preventing the development of interstitial fibrosis after obstructive injury[10].

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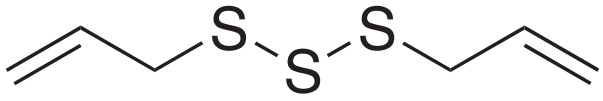
Diallyl Trisulfide

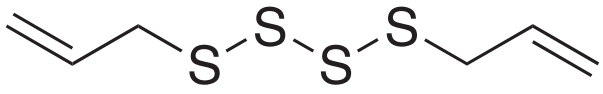
Diallyl Trisulfude (DATS, D3202) and **Diallyl Tetrasulfide (DTS, D3203)** are organosulfur compounds derived from garlic and other *Allium* plants. These compounds are decomposition products of **Allicin (A4440)** that are released when garlic is crushed.

Organosulfur compounds exhibit a variety of biological activities. DATS and DTS play a significant role in garlic's antibacterial activity, inhibiting growth of *Staphylococcus aureus*, *Pseudomonas aeruginosa*, and *Escherichia coli*[1]. In animal models of diabetes, DATS improves cardiac function. This compound increases PI3K-Akt signaling, preventing death receptor- and mitochondria-dependent apoptosis and ameliorating diabetic cardiomyopathy[2].

DATS and DTS also exhibit anticancer activity. Some studies suggest that the antioxidative nature of these compounds is involved in their chemotherapeutic potential[3]. In leukemia cells, DATS increases ROS generation and activates caspases to induce apoptosis and inhibit cell proliferation[4]. In other models, administration of DTS is linked to microtubule depolymerization and G2/M phase cell cycle arrest[5].



D3203 Diallyl Trisulfide



D3203 Diallyl Tetrasulfide

This compound activates JNK signaling, inducing phosphorylation of Bcl-2 and apoptosis in the same study. Additionally, DTS irreversibly inhibits Cdc25, inducing cell cycle arrest and inhibiting proliferation of breast cancer cells[6]. These compounds can also be chemopreventive, suppressing the development of tumors. In animal models of skin tumor carcinogenesis, DATS prevents TPA-induced formation and multiplicity of papillomas[7].

In addition to DATS and DTS, LKT Laboratories also carries **Diallyl Sulfide (D3201), Allyl Disulfide (A4544), L-Deoxyalliin (D1757), L-Alliin (A4444)**, and **L-(+)-Alliin (A4443)**.

References:
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Erucin

Erucin (E6880) is an isothiocyanate found in cruciferous vegetables such as broccoli and arugula. Isothiocyanates are phytochemicals with well known biological applications such as anticancer activity. Other examples of isothiocyanates include **Sulforaphane (S8044)** and **Iberin (I0416)**.

Erucin and sulforaphane display chemopreventive activity, inhibiting development of bladder cancer[1]. This activity is associated with downregulation of survivin, EGFR, and HER2/neu (EGFR2). Erucin also inhibits survival of lung carcinoma cells by activating p53 and p21 signaling and inducing PARP-1 cleavage[2].

The anticancer activity of erucin has also been investigated in prostate cancer models. In prostate adenocarcinoma cells, erucin increases p21 expression and ERK1/2 phosphorylation, suppressing cell growth[3].

Additionally, erucin upregulates activity of phase I and II enzymes, induces apoptosis and cell cycle arrest, and regulates androgen receptor signaling pathways[4].

The ability of erucin to decrease oxidative stress and damage is also beneficial in preventing or delaying pathologies associated with neurodegenerative diseases. By increasing the levels of glutathione and antioxidative enzymes, erucin may show benefit in research models of Parkinson's disease[5].

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E6880 Erucin

Exendin-4 and Alzheimer's Disease

Exendin-4 (E9417) is a peptide that acts as an agonist on the glucagon-like peptide 1 (GLP-1) receptor; it was first found in the venom of *Heloderma suspectum*, the Gila monster. GLP-1 is an endogenous peptide that induces glucose-dependent stimulation of insulin secretion. As an agonist at GLP-1 receptors, exendin-4 also stimulates insulin secretion and exhibits activity in the treatment of diabetes.

Recent research links neurodegenerative diseases such as Alzheimer's disease with dysregulation of brain insulin and insulin-like growth factor (IGF) signaling mechanisms[1]. Reduced levels of insulin and IGF have been found in subjects with Alzheimer's disease or other dementia-associated neurodegenerative diseases. Additionally, diabetes is a known risk factor for Alzheimer's disease.

Exendin-4 may be useful in the treatment of Alzheimer's disease due to its ability to stimulate insulin secretion. GLP-1 and its analogues offer protection to memory formation processes, synapses, and synaptic formation, preventing oxidative stress-induced neuronal damage; additionally, they reduce amyloid-β plaque formation[2]. Exendin-4 is also centrally available as it crosses the blood-brain barrier[3].

In models of Parkinson's disease and cerebral ischemia, exendin-4 protects motor activity. This peptide decreases brain damage and improves outcomes an animal models of transient middle cerebral artery occlusion stroke[4]. The same study shows that exendin-4 protects dopaminergic neurons against degeneration, preserves dopamine levels, and improves motor function in animal models of Parkinson's disease.

LKT Laboratories also carries other GLP-1 receptor agonists, such as **Exendin-3 (E9416)** and **GLP-1, 7-37 (G4482)**.

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Heloderma suspectum

Fluoxetine Hydrochloride

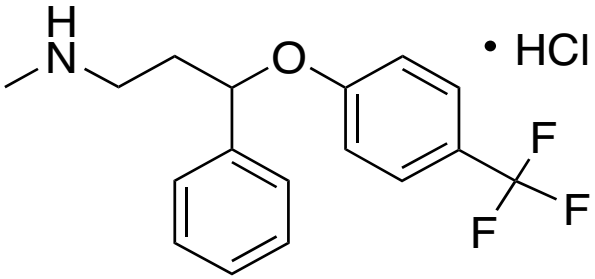
Selective serotonin reuptake inhibitors (SSRIs) increase the level of circulating serotonin in the brain by targeting serotonin transporters and preventing pre-synaptic reuptake. Serotonin is an important neurotransmitter involved in mood regulation, appetite, memory processing, reward, and sleep.

Fluoxetine Hydrochloride (F4780) is an atypical SSRI that also exhibits moderate affinity for norepinephrine transporters, serotonin receptors, and sigma receptors, allowing it to modulate extracellular norepinephrine and dopamine levels as well[1].

Many antidepressants also exhibit analgesic or antinociceptive properties and show activity in models of migraine and neuropathic pain. Fluoxetine-induced antinociception is dependent on modulation of both serotonergic and opioid signaling[2].

Another study analyzed the influence of fluoxetine on kinases involved in intracellular signaling after stimulation with mitogens. Fluoxetine modulates PKC and PKA signaling pathways through calcium mobilization, altering T-cell proliferation[3].

Some antidepressants also exhibit anticancer activity, an added benefit given the co-morbidity of cancers with mood disorders. Fluoxetine itself decreases cell viability and induces apoptosis in hepatocellular carcinoma cells through modulation of MAPK signaling[4].



F 4780 Fluoxetine Hydrochloride

References:
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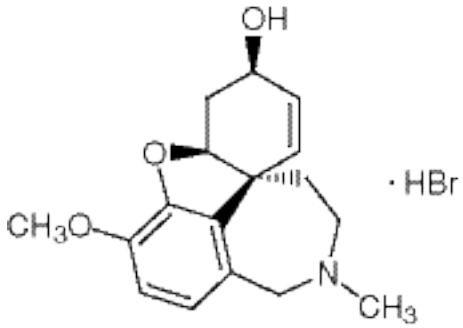
Galantamine Hydrobromide

Galanthus woronowii

Galantamine Hydrobromide (G0246) is an aceylcholinesterase inhibitor and allosteric agonist at nicotinic and muscarinic acetylcholine receptors; it is partially selective for α7 nicotinic acetylcholine receptors. Galantamine is used in the treatment of Alzheimer's disease and offers a broad window of neuroprotection against hypoxia[2].

Galantamine increases acetylcholine levels by inhibiting acetylcholinesterase and increases acetylcholine-mediated signaling through its receptors. This compound increases nicotinic acetylcholine receptor density, enhancing synaptic transmission, cognitive function, and long-term potentiation[3]. Galantamine also promotes hippocampal neurogenesis in animal models in an IGF2-dependent manner[4].

Galantamine exhibits activity in neuro-degenerative diseases such as Alzheimer's disease in other ways as well. This compound decreases oxidative neuronal damage by preventing activation of P2X7 receptors and limits membrane fluidity disturbances[5].



G0246 Galantamine Hydrobromide

References:
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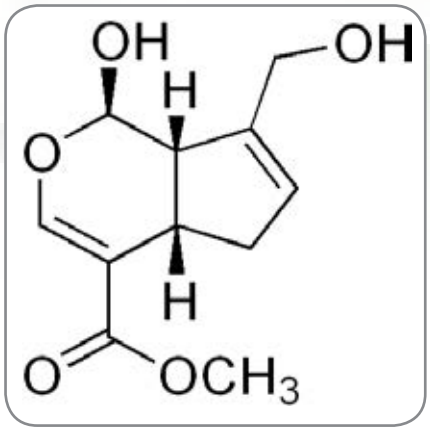
Genipin

Genipin (G1853) is a compound found in the fruit extract of plants in the genus *Gardenia*, a group of flowering evergreen shrubs and small trees. Genipin was initially discovered as a cross-linker for proteins but is now known as an inhibitor of uncoupling protein 2 (UCP2), a mitochondrial carrier protein that negatively regulates glucose-stimulated insulin secretion[1]. When UCP2 is expressed at high levels, insulin secretion is down-regulated; this mechanism likely contributes to the development of type 2 diabetes mellitus[2].



G1853 Genipin

Genipin displays a broad variety of biological activities including neuroprotective effects. Genipin protects hippocampal neurons against amyloid-β toxicity[3]. Amyloid-β oligomer aggregation is a highly neurotoxic hallmark of Alzheimer's disease. Genipin also protects cells from calcimycin-induced cytotoxicity mediated by endoplasmic reticulum stress[4]. This compound may be effective in preventing or limiting the neurodegeneration observed in Alzheimer's disease and Parkinson's disease, both of which involve oxidative stress.

Genipin also exhibits other neuroactive effects. This compound displays antidepressant activity equal to that of **Fluoxetine Hydrochloride (F4780),** a well-established serotonin reuptake inhibitor[5]. In animals undergoing the forced swim and tail suspension tests, genipin improves performance, potentially by increasing levels of serotonin and norepinephrine.

Genepin has been studied extensively for its protein cross-linking activity; new research applications have recently increased interest in this compound. Proteins cross-linked with genipin are currently under investigation in various studies to enhance drug delivery, promote differentiation, and design tissue engineering scaffolds[6-8].

References:
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Compounds found in Green Tea

The extract of green tea leaves contains a variety of compounds with many biological activities. Originally isolated and studied as antioxidants, these compounds have since displayed additional anticancer, antiviral, and anti-inflammatory activities.

The primary active ingredients in green tea extracts are catechins such as **Epigallocatechin Gallate (EGCG, E6234), (-)-Epigallocatechin (EGC, E6233), (-)-Epicatechin Gallate (ECG, E6232)**, and **(-)-Epicatechin (E6231)**. These polyphenols exhibit excellent chemopreventive and chemotherapeutic potential, suppressing cell growth in several different in vitro models of cancer by altering MAPK signaling, CDK expression, topoisomerse I activity, and NF-κB activation[1]. Green tea catechin EGCG suppresses activation of EGFR, IGF-1R, and VEGFR2, inducing apoptosis and inhibiting proliferation of colorectal cancer cells and hepatocellular carcinoma cells[2].

Green tea catechins also display other biological activities. Epicatechin inhibits replication of hepatitis C virus and downregulates expression of COX-2, iNOS, TNF-α, and IL-1β in vitro[3]. In erythrocytes, these compounds prevent t-BHP-induced increases in malondialdehyde and decreases in glutathione[4].

Additional components of green tea extract include flavonoids such as **Quercetin (Q8016), Myricetin (M9367)**, and **L-Theanine (T2816)**. In animal models of subarachnoid hemorrhage, quercetin ameliorates vehavioral deficits by enhancing activity of superoxide dismutase and glutathione peroxidase and decreases levels of malondialdehyde[5]. Myricetin induces ROS-dependent apoptosis in leukemia cells[6]. In spontaneously hypertensive animal models, administration of L-theanine significantly decreases blood pressure[7].

LKT Laboratories carries additional catechins and flavonoids as well as mixtures of the above compounds, such as **Green Tea Polyphenols (G6817)** and a high purity **Catechin Mixture (99%, C0278)**.

References:
1. Lambert JD, Yang CS. J Nutr. 2003 Oct;133(10):3262S-3267S.
2. Shimizu M, Adachi S, Masuda M, et al. Mol Nutr Food Res. 2011 Jun;55(6):832-43.
3. Lin YT, Wu YH, Tseng CK, et al. PLoS One. 2013;8(1):e54466.
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Hedgehog Signaling

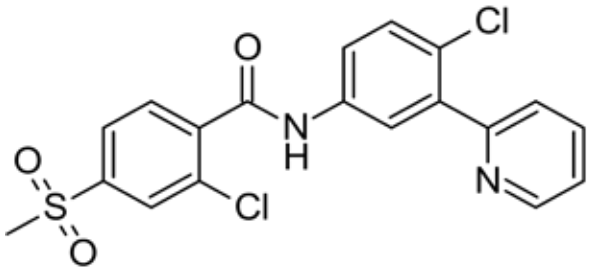
The hedgehog (Hh) signaling pathway is a key regulator of embryonic development in all animals. Hh signaling was first studied in *Drosophila*, where it is necessary in embryogenesis and metamorphosis. Mammals have three Hh homolog proteins: sonic hedgehog (SHH), desert hedgehog (DHH) and Indian hedgehog (IHH).

Out of these three homologs, SHH is the best studied. SHH binds Patched-1, a transmembrane receptor, allowing activation of Smoothened (Smo), a nearby protein. Without SHH, Patched-1 inhibits Smo activity. Activation of Smo results in activation of GLI transcription factors Gli1 and Gli2 (activators) and Gli3 (a repressor). Activated GLI accumulates in the nucleus where it regulates transcription of genes involved in embryogenesis, limb development, adult stem cell proliferation, and hair follicle growth[1].

Alterations in Hh signaling are linked to a variety of diseases. Inhibition of Hh signaling during fetal development causes holoprosencephaly, potentially resulting in cyclopia[2]. Aberrant activation of this pathway is implicated in the development of various cancers likely through transformation of adult stem cells into cancer stem cells[3]. Hh signaling may also play a role in angiogenesis and metastasis[4]. New inhibitors of Hh signaling pathway components are in development as chemotherapeutics to treat a wide range of malignancies.

Hedgehog signaling inhibitors:
C0145 Calcitriol
C8069 Curcumin
C8070 Curcumin (high purity)
F5668 Forskolin
G1652 Genistein
I5034 Imiquimod
R1780 trans-Retinoic acid
V1868 Veratramine

Smoothened modulators:
B1870 Berberine Hydrochloride Hydrate
C9710 Cyclopamine
G1408 GDC-0449 (Vismodegib)
I7870 Itraconazole
J1870 Jervine
N8663 NVP-LDE225 Diphosphate
P8370 Purmorphamine



G1408 GDC-0449 (Vismodegib)

References:
1. Singh BN, Koyano-Nakagawa N, Donaldson A, et al. Genes (Basel). 2015 Jun 23;6(2):417-35.
2. Lee ST, Welch KD, Panter KE, et al. J Agric Food Chem. 2014 Jul 30;62(30):7355-62.
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Histone Deacetylase Inhibitors

Histone deacetylase (HDAC) inhibitors are a class of cytostatic agents that inhibit the proliferation of tumor cells in various models by inducing cell cycle arrest, differentiation, or apoptosis[1].

Cells carry out gene expression by controlling the coiling and uncoiling of DNA around histones. This activity is regulated by histone acetylases, which acetylate lysine residues on each histone, loosening chromatin and allowing space for transcription to occur. Conversely, HDACs remove acetyl groups from the lysine residues, forming a more tightly wound, less active chromatin. Inhibiting HDAC induces the accumulation of hyperacetylated nucleosome core histones in most regions of chromatin and allows transcription of genes. HDAC inhibitors can induce p21 gene expression; p21 is a regulator of tumor suppressor p53 activity. HDAC inhibitors can also modulate expression of retinoblastoma, a protein that suppresses cell proliferation. HDAC inhibitor-induced chromatin inactivation and DNA methylation may inhibit growth and metastasis in centrally-mediated cancers such as glioma[2-4].

LKT Laboratories carries a variety of HDAC inhibitors that are used in anticancer, antiviral, immunomodulatory, anti-inflammatory, and neuroprotective research applications.

References:
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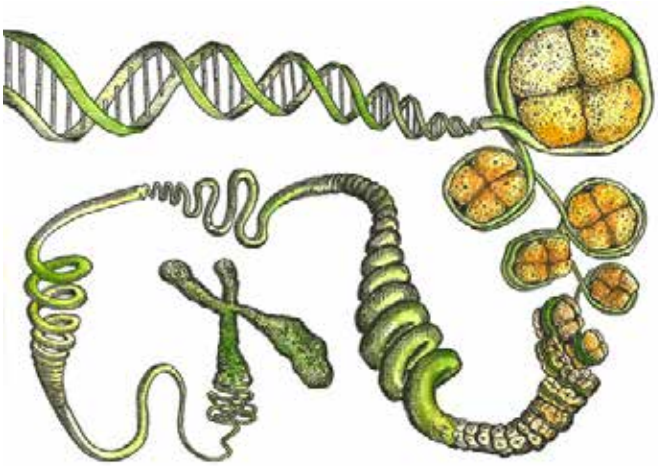
See the representative list below for a selection of HDAC inhibitors.

A6132 Apicidin
B1746 Belinostat
B8275 n-Butyric Acid
C2968 Chrysin
C8112 CUDC-907
E5477 Entinostat
I7559 Isoliquiritigenin
L0528 LBH-589
M2409 MGCD-0103
P2815 Phenylbutyrate Sodium
P2922 Phenylhexyl Isothiocyanate
R5749 Romidepsin
S1069 Scriptaid
B8276 Sodium Butyrate
T6933 Trichostatin A
T8000 Tubacin
T8006 Tubastatin A Hydrochloride
V0147 Valproic Acid Sodium Salt
V5734 Vorinostat



Inhibition of histone deacetylases induce cell cycle arrest by interferring with regular DNA coiling

Isoflavones

Isoflavones are a group of compounds that can be found in plants; many of them exhibit a variety of biological activities, including antioxidative properties. Isoflavones are primarily found in legumes such as soybean, alfalfa, peanut, and red clover. Isoflavones naturally occur as glycosides but can be hydrolyzed to a more bioactive and lipophillic aglycone form.

These compounds display many research applications in cell and animal models, including protection against UV-induced DNA damage[1]. In animal models, isoflavones improve antioxidative capacity and increase levels of estrogen receptors, decreasing incidence and number of mammary tumors[2]. These compounds also exhibit cardioprotective activity, inhibiting the development of cardiac hypertrophy[3]. Additionally, isoflavones prevent bone turnover and maintain bone mineral density in ovariectomized animals, indicating potential as a treatment for osteoporosis[4].

LKT Laboratories carries a wide range of isoflavones, including the representative compounds below.

B3358 Biochanin A
D0033 Daidzin
D0032 Daidzein
E6781 Equol
F5770 Formononetin
G1653 Genistin
G1652 Genistein
G4799 Glycitin
G4798 Glycitein
N0068 Naringenin
P8117 Puerarin

References:
1. Iovine B, Garofalo M, Orefice M, et al. Clin Exp Dermatol. 2014 Apr;39(3):391-4.
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3. Maulik SK, Prabhakar P, Dinda AK, et al. Can J Physiol Pharmacol. 2012 Aug;90(8):1117-25.
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JAK/STAT Inhibitors

Janus kinases (JAKs) and signal transducers and activators of transcription (STATs) are two primary components of a signaling cascade that transmits extracellular messages across the cell membrane and into the nucleus where gene transcription is altered. The JAK/STAT signaling pathway is involved in cell growth, differentiation, survival, and pathogen resistance; components of this pathway are expressed in various cell types but are highly prevalent in immune cells. This pathway mediates the effects of interferons, cytokines, and growth factors. Dysregulation of JAK/STAT signaling is related to the development of many diseases. STATs are transcription factors that binds promotor regions of DNA and induce expression of downstream DNA sequences. In some cancers, STAT3 and STAT5 are constitutively activated, resulting in overactive signaling and unchecked gene transcription[1-2]. JAKs kinases that are activated by transmembrane receptors and are responsible for phosphorylating STATs. Gain-of-function mutations in JAK are linked to hematological malignancies such as thrombocythemia, myelofibrosis, and leukemia[3-4].

Inhibitors of JAK signaling:
A9812 AZD-1480
B5000 BMS-911543
C9876 CYT-387
G4662 GLPG-0634
I5210 INCB18424
J0240 JAK2 Inhibitor V
P3209 Piceatannol
T2404 TG101348
T5720 Tofacitinib Citrate
T9969 Tyrphostin AG490
W2933 WHI-P131 (JANEX-1)

Inhibitors of STAT signaling:
C0171 Carboplatin
C5870 Corosolic Acid
C7097 Cryptotanshinone
D1850 Demethoxycurcumin
E6234 Epigallocatechin Gallate
N0163 2-(1,8-Naphthyridin-2-ly)phenol
S1872 Sesamin
S5868 Sorafenib
S8044 R,S-Sulforaphane
T0154 Tanshinone IIA

References:
1. Yu H, Pardoll D, Jove R. Nat Rev Cancer. 2009 Nov;9(11):798-809.
2.Berger A, Sexl V, Valent P, et al. Oncotarget. 2014 Oct 30;5(20):9564-76.
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LRRK2 Inhibitors

Leucine-rich repeat kinase 2 (LRRK2) is a protein that can be found in the cytoplasm and the mitochondrial outer membrane. LRRK2 gain-of-function mutants are associated with increased risk for Crohn's disease as well as Parkinson's disease. Models of Parkinson's disease show that LRRK2 mutations affect vesicular trafficking, autophagy, protein synthesis, and cytoskeletal function[1].

LRRK2 interacts with Parkin, a ligase involved in protein degradation; mutant forms of Parkin are associated with the development of a juvenille familial form of Parkinson's disease[2]. Expression of LRRK2 mutants results in shortening of dendrites in neurons in vitro[3]. Other mutations induce calcium imbalance, autophagic clearance of mitochondria, and neuro-degeneration and inflammation, all signs of Parkinson's disease[4-5].

Currently, few options are available to slow or prevent the progression of this disease. Although some compounds show activity in research models, the field is still growing. However, inhibition of LRRK2 suppresses its kinase activity, lessening pathologies associated with Parkinson's disease in cellular and animal models.

LKT Laboratories carries several LRRK2 inhibitors that are currently under investigation for their neuroprotective applications in various research models.

PF-06447475 (P2100) suppresses neurodegeneration and inflammation induced by α-synuclein in animal models[5]. In vitro, **CZC-54252 (C9808)** limits mutant LRRK2-induced injury of rodent and human neurons[6]. **GNE-7915 (G5216)** inhibits LRRK2 with high potency across several species[7].

References:
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Memantine

Alzheimer's disease is the most common neurodegenerative disorder and a highly prevalent cause of dementia with aging. Several pathophysiological events are triggered in Alzheimer's disease that ultimately lead to signaling pathway dysfunction, failure of neurotransmission, and neuronal death. **Memantine (M1749)** is used as a treatment to slow the progression of this disease[1].

Several neuroprotective compounds inhibit cholinergic signaling to improve congitive abilities. Memantine instead acts as a noncompetitive antagonist at NMDA receptors, blocking glutamate signaling[2]. Memantine inhibits prolonged influx of calcium ions from extrasynaptic receptors, lessening neuronal excitotoxicity.

Memantine also interacts with a variety of ligand-gated ion channels such as nicotinic acetylcholine receptors (nAChRs), dopamine receptors, and serotonin receptors[3]. Most of this activity does not contribute to the effects of memantine on cognitive function, although it may be related to the antidepressant, antitussive, and antinociceptive activities of memantine[4-6].

At therapeutic concentrations, memantine promotes synaptic plasticity and preserves or enhances memory in animal models of Alzheimer's disease. Additionally, memantine protects against excitotoxic neurodegeneration. Additional research indicates that memantine suppresses toxicity induced by amyloid-β (Aβ) plaque formation, potentially inhibiting the production of Aβ by altering APP processing[7].

References:
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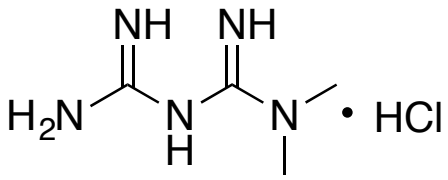
Metformin

Metformin Hydrochloride (M2076) is a compound that has been used for its antidiabetic properties for several decades. Metformin is typically used to reduce blood sugar in subjects with type 2 diabetes mellitus. Although the mechanism of action is not well characterized, metformin is known to decrease hepatic glucose production by inhibiting mitochondrial respiration and activating AMPK[1-2]. Metformin also enhances peripheral glucose uptake and increases insulin sensitivity[3].

More recently, metformin has come back into the research spotlight for other potential applications. Metformin shows activity in cell and animal models of cancer as well as aging.

In various cancer models, metformin stops cell growth, cancer metastasis, and tumorigenesis; these effects are likely due to its ability to limit mitochondrial oxidative phosphorylation. In primary ovarian cancer cells, metformin induces cell cycle arrest and apoptosis[4]. In cell and animal models of cholangiocarcinoma, metformin limits cell cycle progression, halts cell proliferation, and suppresses growth of xenograft tumors[5]. Analysis of several clinical studies focusing on metformin administration in breast cancer subjects indicates a link between metformin use and lower mortality rates[6].

New studies suggest that metformin may also slow down the effects of aging. In *Drosophila* intestinal stem cells, metformin inhibits age- and oxidative stress-induced centrosome amplification, limiting stem cell aging[7]. Metformin also targets complex I of the mitochondrial electron transport chain, altering mitochondrial free radical production[8]. In *Caenorhabditis elegans*, metformin administration extends lifespan through the promotion of mito-hormesis[9]. Additional publications highlight the connection between inactivation of insulin signaling or caloric restriction -- both of which can be achieved with metformin -- and increased life span[10].



M2076 Metformin Hydrochloride

References:
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NSAIDS

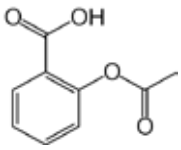
Non-Steroidal Anti-Inflammatory Drugs

Non-steroidal anti-inflammatory drugs (NSAIDs) typically reduce aches, pains, swelling, and fever by inhibiting activity of COX-1 and COX-2. In addition to the analgesic, anti-inflammatory, and anti-pyretic effects, compounds such as **Acetylsalicylic Acid (Aspirin, A0819)** and **Piroxicam (P3269)** also exhibit chemopreventive activity, decreasing the risk of colon cancer, breast cancer, intestinal cancer, and prostate cancer[1-4]. This inhibition of carcinogenesis is likely due to both COX-dependent and COX-independent mechanisms.

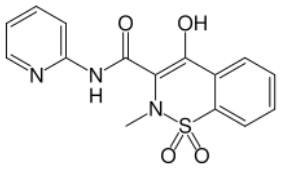
Inhibition of COX enzymes results in an increase in prostaglandin precursor arachidonic acid. In colon tumor cells, arachidonic acid stimulates the conversion of sphingomyelin to ceramide and induces apoptosis[5]. Animal models of colorectal cancer support this finding, showing apoptosis and inhibition of aberrant crypt foci development in animals administered **Sulindac (S8145)**[6].

A separate chemopreventive mechanism includes inhibition of PDE5. Suppression of this enzyme increases levels of cGMP and activates PKG. This results in downstream activation of JNK1 and downregulation of β-catenin-mediated transcription; as a result, cell cycle arrest and apoptosis are induced and proliferation and angiogenesis are inhibited[7-8]. Both sulindac and **Flurbiprofen (F4481)** act through this pathway, preventing development of cancers in cellular models.

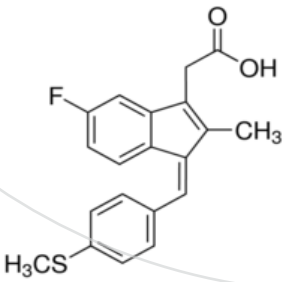
LKT Laboratories carries a wide variety of NSAIDs, including both non-selective COX inhibitors and COX-2 selective inhibitors such as **Etoricoxib (E7858), Meloxicam (M1644)**, and **Celecoxib (C1644)** and many more!



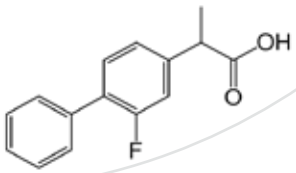
A0819 Acetylsalicylic Acid



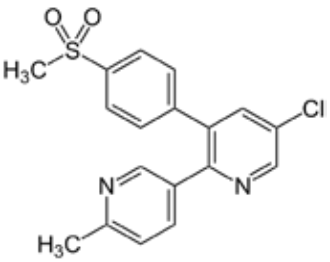
P3269 Piroxicam



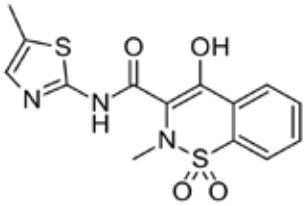
S8145 Sulindac



F4481 Flurbiprofen



E7858 Etoricoxib



M1644 Meloxicam

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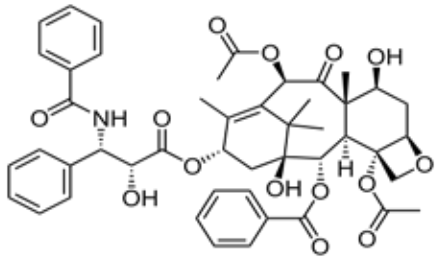
Paclitaxel

The natural anticancer diterpenoid **Paclitaxel (P0092 and P0093)** was discovered in 1971[1]. Paclitaxel was originally developed from the stem bark of the Pacific yew tree (*Taxus brevifolia Nutt*), but has since been developed in a synthetic manner. Paclitaxel exhibits chemotherapeutic activity against various cancer cell lines, including leukemias, sarcomas, and lung tumor cells.

Most antimitotic chemotherapeutic compounds such as **Colchicine (C5645), Vinblastine Sulfate (V3253)**, and **Podophyllotixin (P5712)** inhibit microtubule polymerization[2-4]. Instead, paclitaxel stabilizes assembled microtubules through the binding of β-tubulin, inhibiting microtubule depolymerization and breakdown during cell division. As a result, cells are unable to progress through mitosis and apoptosis is triggered. Paclitaxel is clinically used to treat a variety of cancers, including lung cancer, ovarian cancer, breast cancer, head and neck cancers, and Kaposi's sarcoma[5-8].

LKT Laboratories carries both synthetic and naturally-sourced paclitaxel as well as a variety of other taxanes and intermediates. See the list below for a sample of representative products.

T0093 2'-Acetyltaxol
T0095 Baccatin III
T0100 10-Deacetyltaxol
T0101 7-Epi-10-Deacetyltaxol
T0102 7-Epi-Taxol
T0105 Taxol C
T0106 Xylosyltaxol
D5709 Docetaxel
and many others!



P0092/P0093 Paclitaxel

Taxus brevifolia

References:
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Paroxetine Hydrochloride

Paroxetine Hydrochloride (P0297) is an antidepressant commonly used in the treatment of major depression, obsessive-compulsive disorder, post-traumatic stress disorder, and both social and generalized anxiety. Paroxetine is a selective serotonin reuptake inhibitor (SSRI), that increases extracellular levels of serotonin by inhibiting presynaptic reuptake. As a result, more serotonin is available to bind to the postsynaptic receptor. Serotonin helps regulate mood, appetite, and sleep; it also facilitates some cognitive functions, such as memory and learning.

Paroxetine is a phenylpiperidine derivative, unrelated to tricyclic or tetracyclic compounds traditionally used as antidepressant medication. Those compounds are less selective and exhibit more undesirable side effects than their newer counterparts[1].

Though paroxetine has been used as an antidepressant for over a decade, new studies continue to support its efficacy, revealing new mechanisms of action. Epigenetic processes have been identified as possible crucial regulatory mechanisms in psychiatric disease, and a link between compounds such as paroxetine and epigenetic machinery in the brain are under exploration.

In one study, paroxetine indirectly targets DNA methyltransferase 1 (DNMT1), an enzyme responsible for methylating CpG dinucleotides in mammalian DNA. Paroxetine alters DNMT1 activity through modulation of histone methyltransferase (HMT) G9a, an activator of DNMT1. Administration of paroxetine decreases G9a protein levels and inhibits interactions between G9a and DNMT1[2]. Thus, as paroxetine affects DNMT1 activity via G9a, the connection between small molecules, epigenetics, and psychiatric disease warrants further exploration.

References:
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PARP Inhibitors

Poly (ADP-ribose) polymerase (PARP) inhibitors are a group of compounds used in the treatment of cancer, neurodegenerative diseases, and cardiovascular diseases. PARP is an enzyme responsible for repairing single-stranded DNA breaks, and when inhibited, causes double-stranded DNA breaks to occur during the rapid replication of DNA that is characteristic of many diseases such as cancer[1]. This collection of double-stranded DNA breaks grows rapidly; as the cell is often unable to repair them quickly enough, cell death may occur. Normal cells undergo replication slowly enough that DNA may be repaired by other mechanisms such as homologous repair, allowing them to more easily survive PARP inhibition.

Certain cancers in particular are much more dependent on PARP than regular cells, making them excellent targets for PARP inhibition. In addition to treating breast and pancreatic cancers, PARP inhibitors have shown promise in the treatment of glioma, medulloblastoma, and other brain cancers. **3-Aminobenzamide (A4931)** increases efficacy of co-administered chemotherapeutics in cellular models of glioma[2]. **Velaparib (V1745)** slows tumor progression with combined with **Temozolomide (T1849)**, an alkylating agent, versus temozolomide alone[3]. **Olaparib (O4402)** increases radiation sensitivity in a variety of cell lines, including ependymoma, glioma, and medulloblastoma cells[4].

Other PARP inhibitors carried by LKT Laboratories include **AZD2461 (A9612)** and **PJ34 Hydrochloride (P3600)**.

References:
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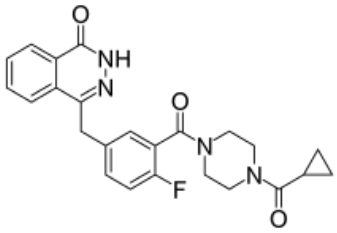

O4402 Olaparib

Pim Kinase Inhibitors

Pim kinases are serine/threonine kinases that play a significant role in cell cycle progression and apoptosis. There are two isoforms of Pim kinase: Pim-1 and Pim-2. Both kinases are expressed in lymphoid cells and are necessary for cytokine-dependent proliferation[1].

Most current research has examined the structure and function of Pim-1, although both are potentially involved in tumorigenesis. Pim-1 activates cell cycle regulator Cdc25, stimulating cell cycle progression. As a result, it may induce unregulated cell growth. Single nucleotide polymorphisms in the gene that encodes Pim-1 have been associated with the presence of diffuse large cell lymphoma and increased risk of lung cancer. Because of its role in cell division, inhibition of Pim-1 shows activity in the treatment of various cancers[2].

AZD-1208 (A9708) is a Pim-1 inhibitor that induces cell cycle arrest and apoptosis in leukemia cells and inhibits phosphorylation of downstream targets such as Bcl-2, 4EBP1, p70S6K, and S6[3].

SMI-4a (S4932) is another inhibitor of Pim-1. SMI-4a limits phosphorylation of eIF4B, suppresses tumor growth, and induces cell cycle arrest and apoptosis in myeloid and lymphoid cells[4].

Proteasome inhibitor **MLN-2238 (M4455)** modulates expression of tumor suppressor miR33b and downregulates Pim-1 activity in multiple myeloma cells[5].

Mitoxantrone Dihydrochloride (M3379) is a DNA intercalator used to treat multiple sclerosis, lymphomas, and leukemias. Research shows it likely derives some activity from the direct inhibition of Pim-1[6].

References:
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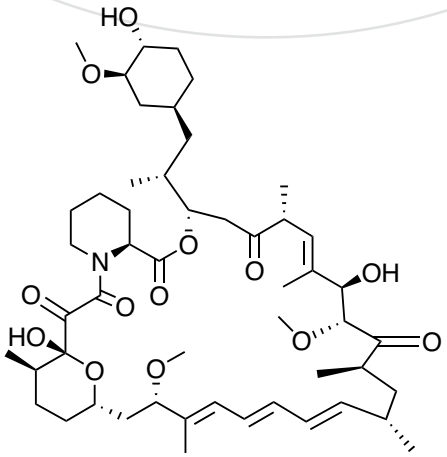
Rapamycin

Rapamycin (R0161) is an immunosuppressant traditionally used to prevent transplant rejection in organ recipients. Rapamycin suppresses mTOR signaling by complexing with FK-binding protein 12 (FKBP12) and preventing mTOR activation[1]. As a result, IL-2 signal transduction is limited and B and T cell activation is inhibited.

Rapamycin also exhibits antiproliferative effects in cancer models. Proliferation of osteosarcoma cells is inhibited by rapamycin. In these cells, rapamycin induced G1 phase cell cycle arrest and autophagy; in paired animal models, it decreased growth of xenograft tumors[2]. In breast cancer cells, this compound induced G1 phase cell cycle arrest, autophagy, and apoptosis[3]. When combined with **Vismodegib (GDC-0449, G1408)**, rapamycin exhibited anticancer activity in cell and animal models of biliary tract cancer. This combination decreased cell viability and proliferation and limited tumor growth[4]. In a clinical setting, rapamycin also displays chemotherapeutic efficacy. This compound decreased visceral tumor volume in subjects with tuberous sclerosis complex skin tumors[5].

More recently, rapamycin has shown activity in models of aging and aging-related disorders. Administration of rapamycin to yeast increased chronological life span[6]. Rapamycin's inhibition of mTOR signaling is likely responsible for the increased accumulation of storage carbohydrates and enhanced stress resistance that was linked to the increase in life span. In genetically heterogeneous male and female animal models, this compound extended median and maximal life span when administered midway through or near the end of the average murine life span[7]. Because mTOR signaling is linked to oxidative stress, the ability of rapamycin to suppress that signaling pathway and decrease oxidative stress is likely a significant factor in its ability to slow development of various diseases and, as a result, increase overall life span[8].



R0161 Rapamycin

References:
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Resveratrol

Phytoalexins are organic metabolites produced by plants in response to fungal infections, heavy metals, or UV radiation[1]. **Resveratrol (R1776)** is a stilbene-type phytoalexin found in the skin and seeds of grapes as well as many other plants such as peanuts, soybeans, and mulberries. Like other phytoalexins, resveratrol exhibits a variety of biological activities, including antioxidative, anticancer, neuroprotective, and cardioprotective effects.

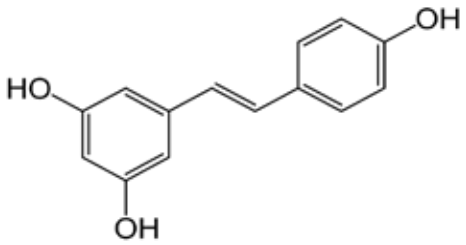
A major dietary source of resveratrol is red wine. Many of the health benefits of red wine consumption have been attributed to the polyphenol fraction, which contains resveratrol. Resveratrol's activation of Sirtuin-1 has been linked to its potential anti-aging and metabolic properties[2].

Resveratrol is a strong antioxidant, inhibiting low-density lipoprotein oxidation and ameliorating oxidative stress in vitro[3-4]. Additional activities of resveratrol include lowering hepatic fat content, reducing cholesterol levels, and inhibiting platelet aggregation[5]. Resveratrol also displays a broad range of chemopreventive and chemotherapeutic activities. This compound exhibits anticancer effects in several cellular and animal models[6]. In lung cancer cells, resveratrol induces caspase-mediated apoptosis; in a similar study in animal models, it inhibits growth of lung cancer tumor xenografts[7]. Resveratrol also inhibits the activities of COX and hydroperoxidase, two enzymes implicated in cancer progression.

LKT Laboratories carries several derivatives of resveratrol that exhibit similar activites. **Trans-3,4',5-Trimethoxy-stilbene (T7134)** is the permethylated derivative of resveratrol; it is more effective than resveratrol in inhibition of cell and tumor growth in models of colorectal carcinoma and prostate cancer[8]. **Triacetyl Resveratrol (T7132)**, a resveratrol prodrug, exhibits improved bioavailability over resveratrol. Like its parent compound, triacetyl resveratrol also exhibits potential anticancer activity[9].

Related Products:

R1776 Resveratrol
P3209 Piceatannol
T7132 Triacetyl Resveratrol
T7134 Trans-3,4',5-Trimethoxy-stilbene
D3331 α,β-Dihydroresveratrol
P7718 Pterostilbene



R1776 Resveratrol

References:
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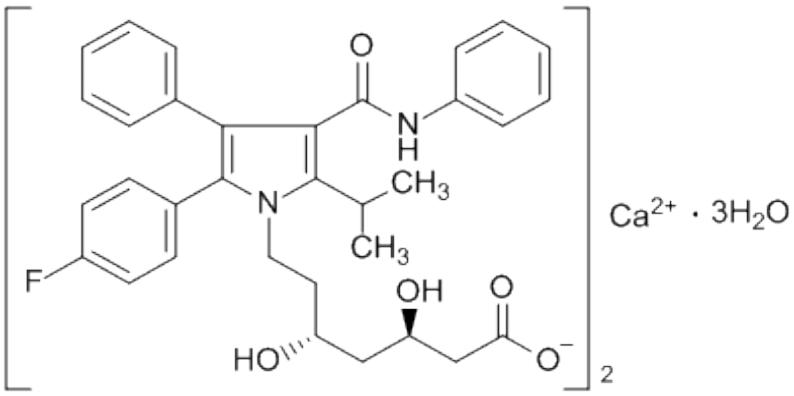
Statins

Antihyperlipidemics
Anticancer Agents

Statins are a group of compounds that exhibit anti-hyperlipidemic activity and are clinically used to lower plasma levels of LDL and total cholesterol[1]. These compounds typically inhibit HMG-CoA reductase, suppressing the production of cholesterol precursor mevalonate[2]. Not only do statins inhibit cholesterol synthesis, they also prevent the production of prenylated proteins, improving endothelial function, inflammatory responses, and cardiac health[3-4].

There are two main groups of statins, one of which includes fungal metabolites and their synthetic analogs such as **Mevinolin/Lovastatin (M1678), Simvastatin (S3449)**, and **Pravastatin Sodium (P6801)**. The other category includes purely synthetic statins such as **Atorvastatin Calcium Trihydrate (A7658), Fluvastatin Sodium (F4482)**, and **Rosuvastatin Calcium (R5974)**[5-6].

Additionally, some statins also exhibit anticancer effects[7]. In myeloid leukemia and medulloblastoma cells, lovastatin induces G1 phase cell cycle arrest and apoptosis, inhibiting cell proliferation[8-9]. In animal models, lovastatin displays anti-angiogenic activity as well, suppressing VEGF secretion and inhibiting tumor-induced vessel formation[10]. In other animal models, rosuvastatin and fluvastatin inhibit Ras protein translocation and pancreatic tumor growth[11].



A7658 Atorvastatin Calcium Trihydrate

Also Available:

C1668 Cerivastatin Sodium
F4482 Fluvastatin Sodium
M1687 Mevinolin/Lovastatin
M1685 Mevastatin
P3576 Pitavastatin Calcium
P6801 Pravastatin Sodium
P6800 Pravastatin Lactone
R5974 Rosuvastatin Calcium
S3449 Simvastatin
S3450 Simvastatin Sodium

References:
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Sulforaphane

Sulforaphane is a cancer chemopreventive agent that belongs to a class of plant-based products that contain isothiocyanate moieties. Isothiocyanates can be found in cruciferous vegetables such as broccoli, cabbage, and arugula[1-2]. Sulforaphane, also known as 4-methylsulfinylbutyl isothiocyanate, exhibits many bioactive properties, including antimicrobial, antioxidative, and anticancer activities. Synthetic **R,S-Sulforaphane (S8044)** is an effective chemopreventive agent; it prevents the development and growth of mammary tumors in animal models[3]. Naturally-occurring isomer **R-Sulforaphane (S8046)** is optically active. Much of sulforaphane's anticancer effect occurs through activation of phase II detoxifying enzymes.

Sulforaphane is an inducer of phase II enzymes such as glutathione-S-transferase and quinone reductase[4-5]. Sulforaphane increases activity and expression of these enzymes as well as γ-glutamyl-transpeptidase in lymphoblastoid cells and prostate cancer cells, inducing apoptosis and inhibiting cell growth[6-7].

References:
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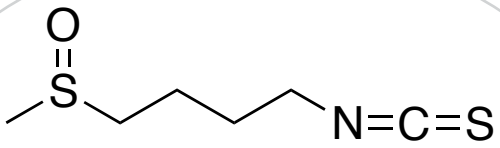
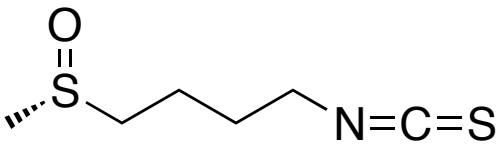
Sulforaphane also induces apoptosis in other cancer cells lines. In colon carcinoma cells, this compound increases expression of Bax and induces release of cytochrome C and cleavage of PARP, resulting in cell cycle arrest and apoptosis[8]. In melanoma cells, sulforaphane increases activation of caspases, Bax, and p53 and decreases expression of Bcl-2, NF-κB, caspase 8, and Bid[9]. These signaling modifications result in apoptosis and inhibition of cell proliferation.

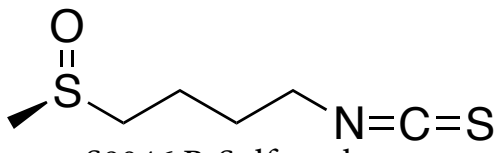
Other mechanisms that contribute to the anticancer effects of sulforaphane are currently under investigation. One such mechanism focuses on the modulation of epigenetic markers. In colon cancer cells, sulforaphane inhibits activity and increases turnover of histone deacetylases[10]. In this study, this compound enhances acetylation and degradation of DNA repair enzymes, preventing them from mending double-stranded DNA breaks; this activity induces cell cycle arrest, autophagy, and apoptosis.



S8044 R,S-Sulforaphane



S8045 S-Sulforaphane



S8046 R-Sulforaphane

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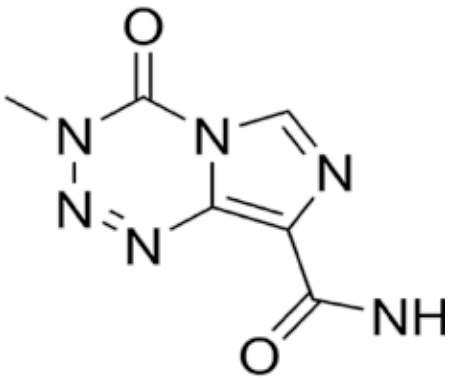
Temozolomide
and MGMT Inhibitors

Temozolomide (T1849) is an alkylating agent used in the treatment of cancers. Like other alkylating agents, temozolomide attaches an alkyl group to DNA at N7 and O6 positions of guanine bases and at the N3 position of adenine bases, interfering with DNA replication[1]. Because cancer cells generally proliferate faster and with less error correcting than normal cells, cancer cells are more susceptible to the effects of these compounds.

Temozolomide has been well studied as a stand-alone therapy and is used to treat oligodendrocytoma, melanoma, and glioblastoma multiforme. Recently, this compound has been studied as a combination therapy. Research suggests additional benefit when temozolomide is co-administered with inhibitors of *O6*-methylguanine DNA methyltransferase (MGMT), an enzyme that repairs alkylations. Compounds that inhibit MGMT include **Lomeguatrib (L5750)** and ***O6*-Benzylguanine (B1855)**[2-3]. In models of glioblastoma, the combination of temozolomide and green tea component **Epigallocatechin Gallate (EGCG, E6234)** improves survival time compared to temozolomide alone[4]. EGCG's inhibition of endoplasmic reticulum chaperone GRP78 is thought to contribute significantly to its anticancer efficacy when administered as a combination therapy with other treatments.



T1849 Temozolomide

References:
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Toll-like Receptor Modulators

Toll-like receptors (TLRs) are pattern recognition receptors expressed in immune cells such as macrophages and dendritic cells; they play a significant role in the development of the innate immune response.

These receptors recognize structurally conserved molecules shared by classes of pathogens. These structures are pathogen-associated molecular patterns. Although associated with pathogens, some structures can also be found endogenously. There are 11 isoforms of TLRs, and each one recognizes distinct ligands, including single-stranded RNA, double-stranded RNA, CpG DNA, lipopolysaccharides, and bacterial peptidoglycans[1].

TLRs are heavily involved in propagation of signals for inflammation, phagocytosis, antigen presentation, and other immune responses, making them good targets for compounds that regulate allergic reactions, inflammation, and autoimmune diseases.

References:
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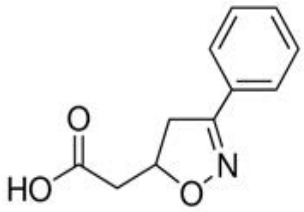
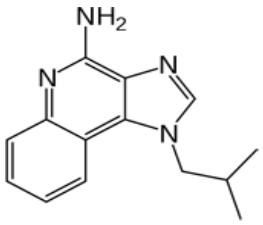
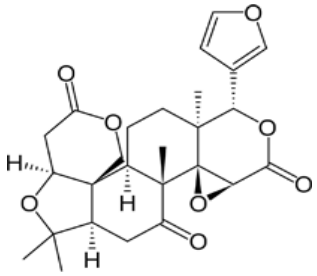


VGX-1027 (V2792) is an inhibitor of TLR4, a receptor that recognizes many endogenous structures. This compound inhibits antigen presentation in models of systemic lupus erythmatosus[2].



Imiquimod (I5034) is an agonist at TLR7 and TLR8 receptors. It recognizes viral RNA sequences, stimulating a Th1-based immune response against Japanese encephalitis virus[3].



Limonin (L3550) is a product that can be isolated from citrus fruits that downregulates expression of TLR2 and TLR, suppressing proinflammatory cytokine release[4].

TRP Channel Modulators

Transient receptor potential (TRP) channels are ion channels located on the plasma membrane of many different cell types. TRP channels play a significant role in sensory transduction, pain signal relay, temperature, taste, and pressure.

TRP vanilloid (TRPV) channels are the most well-studied of these channels. **Capsaicin (C0266)** and **Piperine (P3465)** are TRPV channel activators found in plant sources and are responsible for the hot or spicy flavor of some peppers[1].

TRP canonical (TRPC) channels are often found on cardiomyocytes and nerves; TRPC channels play a role in cardiac hypertrophy[2]. Amyotrophic lateral sclerosis (ALS) treatment **Riluzole (R3347)** activates TRPC5 channels. **Clemizole (C4417)**, a NS4B and histamine receptor blocker, inhibits TRPC5 channels, potentially regulating neurite length[3].

TRP melastatin-like (TRPM) channels are involved in temperature and taste transduction as well as cell adhesion. **Icillin (I0933)** activates TRPM8 channels and inhibits TRPV3 channels, acting as a cooling agent[4]. Other TRP channels include TRP Ankyrin (TRPA) channels, TRP polycystin (TRPP) channels and TRP mucolipin (TRPML) channels.

References:
1. Kissin I. Anesth Analg. 2008 Jul;107(1):271-81.
2. Bush EW, Hood DB, Papst PJet al. J Biol Chem. 2006 Nov 3;281(44):33487-96.
3. Richter JM, Schaefer M, Hill K. Mol Pharmacol. 2014 Nov;86(5):514-21.
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Representative Compounds and Targets:

TRPV
A7085 Arvanil: TRPV1 activator
A9818 Azalastine: TRPV1 activator
C0266 Capsaicin: TRPV activator
C0368 Carveol: TRPV3 activator
G3252 6-Gingerol: TRPV1 activator
M1979 Methyl Salicylate: TRPV1 activator/blocker
N1755 Neomycin Sulfate: TRPV1 blocker
P1634 Peimine: TRPV1 and TRPA1 blocker
P1635 Peiminine: TRPV1 and TRPA1 blocker
P3465 Piperine: TRPV activator
R1774 Resiniferatoxin: TRPV activator
S8151 Sumatriptan: TRPV1 blocker
T6902 Tranilast: TRPV2 blocker

TRPC
C4417 Clemizole: TRPC5 blocker
F4483 Flufenamic Acid: TRPC3 and TRPM2 blocker
N5311 Nocistatin: TRPC activator
R3347 Riluzole: TRPC5 activator

TRPM
G3456 Ginsenoside Rd: TRPM7 blocker
I0933 Icillin: TRPM8 activator, TRPV3 blocker
P7023 Pregnenolone: TRPM3 activator

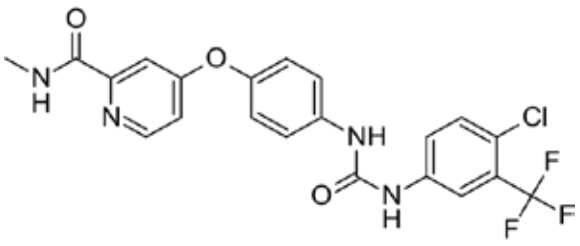
TRPA
A0817 1'-Acetoxychavicol Acetate: TRPA activator
E7556 Etodolac: TRPA activator
P0270 Parthenolide: TRPA activator

VEGFR Inhibitors

Vascular endothelial growth factor receptor (VEGFR) inhibitors are a family of compounds that suppress signaling by VEGF receptor tyrosine kinases. VEGFR is an important signaling mediator involved in angiogenesis, a key process in tumor growth, making this pathway a major target in cancer research. VEGFR recruits circulating endothelial precursor cells and acts as a survival factor for immature tumor blood vessels; blocking this pathway inhibits the development of these vessels, preventing them from activating downstream pathways[1].

Representative VEGFR inhibitors:

A0025 17-AAG
A6818 Arenobufagin
A9435 Axitinib
B5074 BMS-599626
C0006 Cabozantanib
C1613 Cediranib
F5968 Foretinib
I7559 Isoliquiritigenin
M5876 Motesanib
N8460 NVP-BHG712
P0397 Pazopanib
P6002 PP-121
R0020 RAF265
R1626 Regorafenib Monohydrate
S4244 SKLB 610
S5868 Sorafenib
S8098 SU-1498
S8253 Sunitinib Malate
T3585 Tivozanib
V0352 Vandetanib
V0376 Vatalanib Dihydrochloride



S5868 Sorafenib

References:
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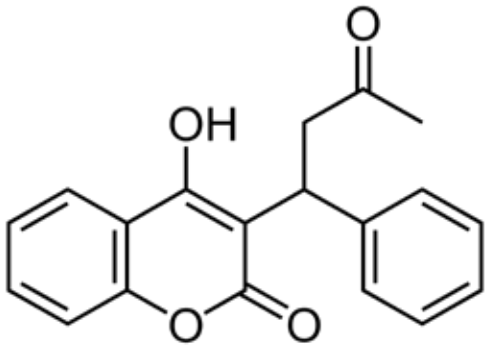
Warfarin

Available forms of warfarin:

W0269 (±)-Warfarin
W0272 S-(-)-Warfarin (>99% ee)
W0273 R-(+)-Warfarin (>99% ee)
W0270 (±)-Warfarin Sodium Clathrate
W0274 S-(-)-Warfarin Sodium (>99% ee)
W0275 R-(+)-Warfarin Sodium (>99% ee)



W0269 Warfarin

(±)-Warfarin (W0269) is an anticoagulant used to prevent the formation and migration of blood clots in blood vessels. Warfarin is one of the most commonly used anticoagulant compounds in clinical practice. Warfarin prevents blood clot formation by inhibiting subunit 1 of the vitamin K epoxide reductase complex[1]. As this enzyme is responsible for recycling oxidized vitamin K to its reduced form to stimulate signaling pathways involved in coagulation, inhibition of vitamin K epoxide reductase lower functional vitamin K levels and prevents the formation of coagulation factors II, VII, IX, and X.

LKT Laboratories carries racemic warfarin as well as optically active enantiomers **S-(-)-Warfarin (W0272)** and **R-(+)-Warfarin (W0273)**, both >99% ee. The two enantiomers are metabolized differently and yield different potencies; the S-enantiomer displays higher efficacy in the prevention of coagulation by a 5:1 ratio[2]. LKT Laboratories also carries the salt forms of these compounds, including **(±)-Warfarin Sodium Clathrate (W0269)**, **S-(-)-Warfarin Sodium (W0272)**, and **R-(+)-Warfarin Sodium**.

Warfarin has several shortcomings as an anticoagulant. The onset of action for warfarin is relatively slow and requires 1-2 days before effects are observed. This compound is sensitive to a wide variety of genetic polymorphisms and is metabolized by cytochrome P450 2C9; it also interacts with many other medications and foods[3]. As a result, dosage is highly variable among subjects, requiring diligent activity monitoring on an individual basis.

References:
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2. Hirsh J, Fuster V, et al. J Am Coll Cardiol. 2003 May 7;41(9):1633-52.
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Isothiocyanates
from Watercress

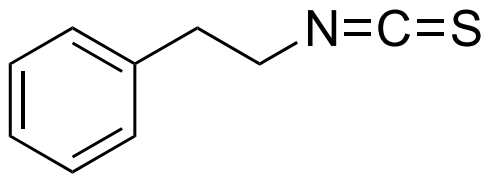
Watercress (*Nasturtium officinale*) is a member of the cruciferous vegetable family. Like other cruciferous plants, watercress is a rich source of isothiocyanates. These compounds exhibit anti-cancer properties and induce activity of phase II enzymes[1]. The primary active component of watercress is **Phenylethyl Isothiocyanate (PEITC, P2508)**, followed by **7-Methylsulfinylheptyl Isothiocyanate (MSH, I7456)** and **8-Methyl-sulfinyloctyl Isothiocyanate (MSO, I7459)**.

PEITC exhibits chemopreventive and chemotherapeutic activities in various models. In prostate cancer cells, PEITC induces apoptotic cell death in a ROS-dependent manner involving inhibition of oxidative phosphorylation and depletion of ATP[2]. In animals with prostate cancer tumor xenografts, PEITC inhibits tumor PECAM-1 and several integrin family proteins, suppressing angiogenesis and tumor growth[3].

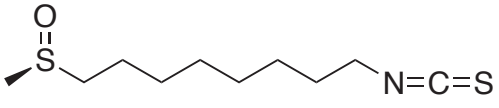
One enzyme that PEITC induces is quinone reductase, a key component in the regulation of ROS levels. Both MSH and MSO also induce quinone reductase activity as well and may exert more potent activity on phase II enzyme induction.

Many other isothiocyanates can be found in cruciferous vegetables, including **R-Sulforaphane (S3046), Phenylhexyl Isothiocyanate (PHITC, P2922)**, and **R-(-)-Iberin (I0417)**. Isothiocyanates that can be found in broccoli and watercress suppress TPA-induced activity of MMP-9 and invasiveness of breast cancer cells in vitro[4]. A separate study indicates that isothiocyanates such as PEITC and sulforaphane directly interact with histones and chromatin remodeling proteins, potentially inhibiting histone deacetylase activity and DNA methylation[5].

LKT Laboratories also offers several of the methylsulfinylalkyl isothiocyanates in chiral form, including **(R)-1-Isothiocyanato-8-(methylsulfinyl)-octane (I7460)** and **(R)-1-Isothiocyanato-7-(methylsulfinyl)-heptane (I7455)**.



P2508 Phenylethyl Isothiocyanate



I7460 (R)-1-Isothiocyanato-8-(methylsulfinyl)-octane

References:
1. Rose P, Faulkner K, Williamson G, et al. Carcinogenesis. 2000 Nov;21(11):1983-8.
2. Xiao D, Powolny AA, Moura MB, et al. J Biol Chem. 2010 Aug 20;285(34):26558-69.
3. Hudson TS, Perkins SN, Hursting SD, et al. Int J Oncol. 2012 Apr;40(4):1113-21.
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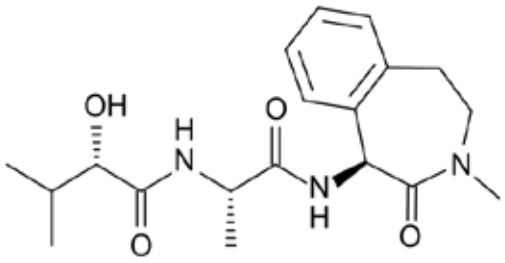
γ-Secretase Inhibitors

γ-Secretase is a multi-subunit protein responsible for cleaving transmembrane proteins such as amyloid precursor protein and Notch. Cleavage of amyloid precursor protein eventually results in the formation of amyloid-β, the main component of amyloid plaques characteristic of Alzheimer's disease[1]. Cleavage of Notch allows for gene transcription and other downstream signal transduction necessary for cell-cell communications involved in embryogenesis, cell differentiation, endocrine development, and potentially tumorigenesis[2]. Notch activity promotes cell survival and suppresses apoptosis.

Available inhibitors:
D0260 DAPT
D1773 Deshydroxy LY-411575
F4432 FLI-06
L9701 LY-450139
M4200 MK-0752



L9701 LY-450139

Inhibition of γ-secretase and Notch signaling can decrease production of amyloid-β peptides and halt or slow cell division, particularly for stem cells. Inhibition of Notch signaling appears to target cancer stem cells, stopping growth and inducing differentiation or apoptosis[3]. Because Notch interacts with many other signaling pathways and components such as PI3K/Akt, Ras, NF-kB, EGFR, and VEGFR, these downstream targets are affected as well.

LKT Laboratories carries several γ-secretase inhibitors that exhibit activity in a variety of research applications. In animal models of Alzheimer's disease, **LY-450139 (L9701)** prevents production of new amyloid plaques[4]. **MK-0752 (M4200)** displays potential as a treatment for brain and CNS-centric cancers[5-6]. **FLI-06 (F4432)** inhibits protein secretion prior to endoplasmic reticulum exit, exhibiting neuroprotective benefit[7].

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2. Artavanis-Tsakonas S, Rand MD, Lake RJ. Science. 1999 Apr 30;284(5415):770-6.
3. Purow B. Adv Exp Med Biol. 2012; 727: 305–319.
4. Beggiato S, Giuliani A, Sivilia S, et al. Neuroscience. 2014 Apr 25;266:13-22.
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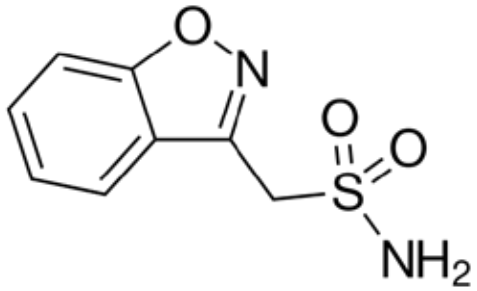
Zonisamide

Zonisamide (Z5653) is an anticonvulsant that is used to treat partial-onset seizures, mixed seizure types of Lennox–Gastaut syndrome, myoclonic seizures, and generalized tonic clonic seizures. Zonisamide inhibits voltage-dependent sodium channels, T-type calcium channels, and carbonic anhydrase. It suppresses the development of neuronal hypersynchronization, a state of brain activity associated with epileptiform seizures[1]. This compound also modulates GABAergic and glutamatergic neurotransmission[1].

Zonisamide exhibits biological activity in many other research applications beyond epilepsy and seizure development. In animal models of Parkinson's disease, this compound reduces nigrostriatal dopaminergic cell death through brain-derived neurotrophic factor signaling and improves survival of dopaminergic neurons and motor function[2].



Z5653 Zonisamide

In animal models of chronic constrictive injury-induced neuropathic pain, zonisamide increases latency to paw withdrawal, indicating analgesic or antinociceptive activity. This occurs in models of chemical, mechanical, and thermal hyperalgesia and allodynia[3].

Zonisamide is also under investigation for its anti-obesity characteristics. In clinical settings, zonisamide improves weight loss[4]. This compound also prevents weight gain, hyperphagia, and elevation in blood glucose levels in animals co-administered second-generation antipsychotics associated with weight gain, such as olanzapine[5].

References:
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