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

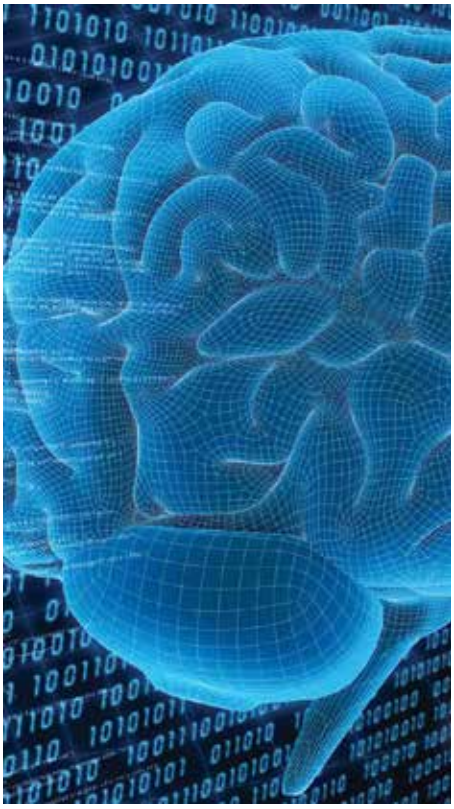
Glioma chemotherapeutics
Glioma chemotherapeutics show pre-clinical and clinical benefit in the treatment of centrally-located or cranial cancers such as oligodendrocytoma and glioblastoma multiforme. These compounds act on a wide variety of targets, such as receptor tyrosine kinases, serine/threonine 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.

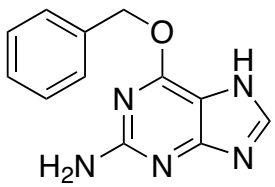
Glioma Chemotherapeutics



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].

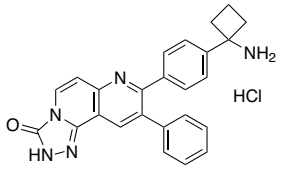
1. Quinn JA, et al. Clin Cancer Res. 2009 Feb 1;15(3):1064-8.
2. Quinn JA, et al. J Clin Oncol. 2009 Mar 10;27(8):1262-7.
3. Qian L, et al. Biomaterials. 2013 Nov;34(35):8968-78.
4. Friedman HS. Clin Cancer Res. 2000 Aug;6(8):2967-8.



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%

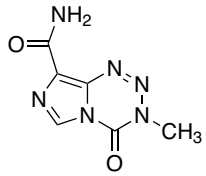
MK-2206



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.

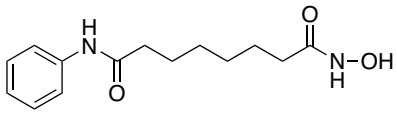
Temozolomide



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].

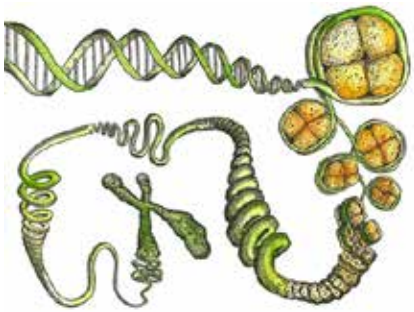
1. Nagasawa DT, et al. Neurosurg Clin N Am. 2012 Apr;23(2):307-22, ix.
2. Wesolowski JR, et al. AJNR Am J Neuroradiol. 2010 Sep;31(8):1383-4.
3. Friedman HS, et al. Clin Cancer Res. 2000 Jul;6(7):2585-97.

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].

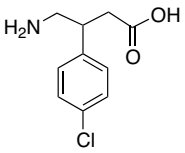
1. Silva G, et al. PLoS One. 2013;8(1):e53766.
2. Xu J, et al. J Neurooncol. 2011 Nov;105(2):241-51.
3. Asklund T, et al. Anticancer Res. 2012 Jul;32(7):2407-13.
4. Lee EQ, et al. Clin Cancer Res. 2012 Nov 1;18(21):6032-9.
5. Matsumoto Y, et al. Psychopharmacology (Berl). 2013 Sep;229(1):51-62.
6. Archin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



Neurotransmitter Modulators

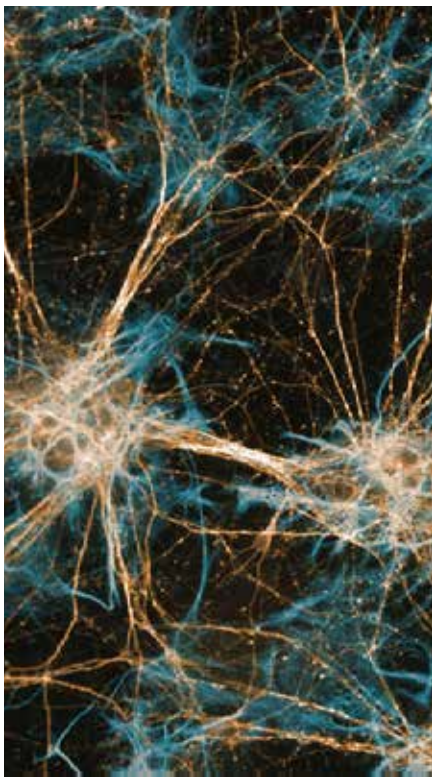
Cat #	Product Name	Description	Purity
A4802	Amantadine HCL	Inhibits α_7 nAChR, NMDAR, MAOB, viral protein M2; potentiates DA release	≥96%
A5234	Amisulpride	Inhibits $D_{2/3}R$, 5-$HT_{2B/7}R$; activates GHBR	≥98%
A5235	Amitriptyline HCL	Inhibits SERT, NET, $D_{1/3/5}R$, 5-$HT_{2/3/6/7}R$, $H_{1/4}R$, $\alpha_{1/2}R$, $M_{1\text{-}5}R$, 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.

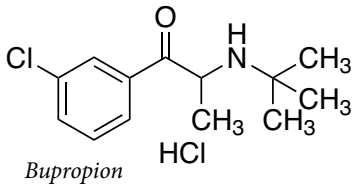


Neurotransmitter Modulators

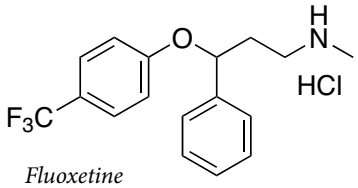
Bupropion

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].

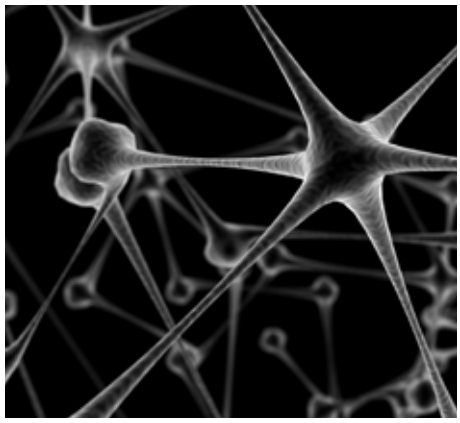
1. Li Z, et al. Ann Intern Med. 2005 Apr 5;142(7):532-46.
2. Cantwell DP. J Clin Psychiatry. 1998;59 Suppl 4:92-4.
3. Shah TH, et al. Am J Hosp Palliat Care. 2010 Aug;27(5):333-6.
4. Dong J, et al. Psychopharmacology (Berl). 2001 Apr;155(1):52-7.
5. Arias HR, et al. Int J Biochem Cell Biol. 2009 Nov;41(11):2098-108.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



Bupropion



Fluoxetine



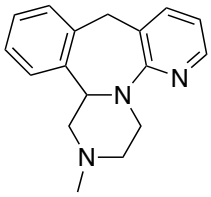
Fluoxetine

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].

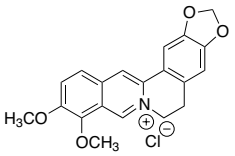
This compound also shows efficacy in altering behaviors associated with autism spectrum disorder as well as pervasive developmental disorder[6].

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].


California Poppy

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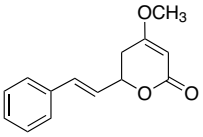
Kavalactones/Kawain



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].


Kava plant

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3. Wruck CJ, et al. Mol Pharmacol. 2008 Jun;73(6):1785-95.
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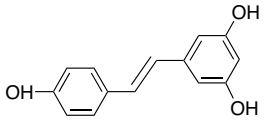
Resveratrol

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].



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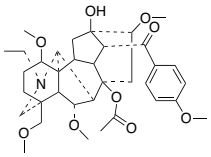
Ion Channel Modulators

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.
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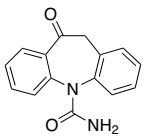
Oxcarbazapine



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].

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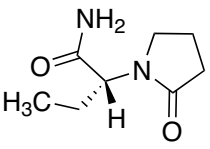
Levetiracetam



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].

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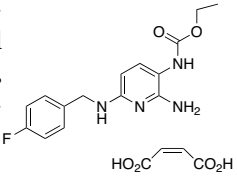
Flupirtine Maleate

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].



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