Web: www.taiclone.com
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Latrepirdine (dihydrochloride)

Catalog No: tcsc5771

Available Sizes

Size: 2mg

Size: 5mg

Size: 10mg

Size: 25mg

Size: 50mg

Size: 100mg

Size: 200mg

Specifications

CAS No:
97657-92-6

Formula:
$C_{21}H_{27}Cl_2N_3$

Pathway:
Neuronal Signaling;Immunology/Inflammation;GPCR/G Protein;GPCR/G Protein;Autophagy;Neuronal Signaling;GPCR/G Protein

Target:
Amyloid-β;Histamine Receptor;Histamine Receptor;Adrenergic Receptor;Autophagy;5-HT Receptor;5-HT Receptor

Purity / Grade:
>98%

Solubility:
DMSO : 6.4 mg/mL (16.31 mM; Need warming)

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Alternative Names:
Dimebolin dihydrochloride

Observed Molecular Weight:
392.37

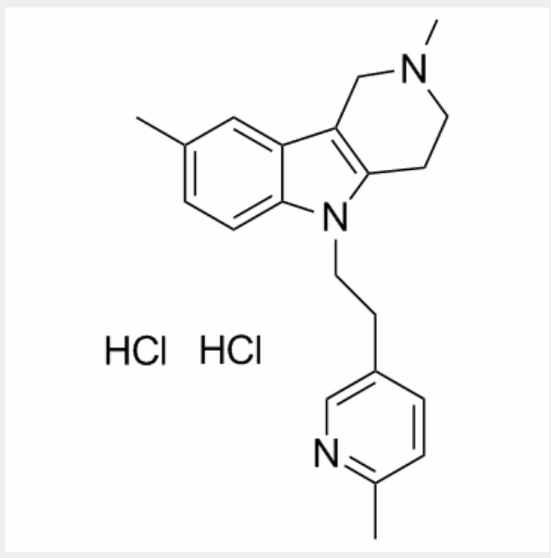
Product Description

Latrepirdine dihydrochloride is a neuroactive compound with antagonist activity at histaminergic, α-adrenergic, and serotonergic receptors. Latrepirdine stimulates amyloid precursor protein (APP) catabolism and **amyloid-β** (**Aβ**) secretion.

IC50 & Target: Amyloid-β (Aβ), Histaminergic receptor, α-adrenergic receptor, Serotonergic receptor[1]

In Vitro: Latrepirdine has been reported to possess several properties that are potentially relevant to the treatment of neurodegenerative diseases: (1) protection of cultured cells from the cytotoxicity of amyloid-β (Aβ) peptide; (2) stabilization of mitochondrial function and calcium homeostasis; (3) modulation of Aβ release from cultured cells, isolated intact nerve terminals, and from hippocampal neurons in living mouse brain; and (4) promotion of neurogenesis in the murine hippocampus. Treatment of cultured mammalian cells with Latrepirdine leads to enhanced mTOR- and Atg5-dependent autophagy. Latrepirdine modulates Atg5-dependent autophagic activity in a dose-dependent manner and via the mTOR-signaling pathway. HeLa cells stably expressing LC3 fused are treated with EGFP (eGFP-LC3) for 3 or 6 hours in the absence or presence of 50 μM Latrepirdine. Treatment with Latrepirdine for 3 or 6 hours markedly enhances the number of eGFP-LC3 punctae, indicating that Latrepirdine induces formation of autophagosomes. Next, mouse N2a neuroblastoma cells are treated in the absence (vehicle) or presence of 5 nM, 500 nM or 50 μM Latrepirdine for 3 or 6 hours in order to determine the effects of acute drug treatment on the regulation of autophagy. A significant and dose-dependent increase is observed in LC3-II levels in N2a cells following 3- or 6-hour treatment with either 500 nM or 50 μM Latrepirdine. A significant decrease of p-mTOR and p-S6K from N2a cells treated with 50 μM Latrepirdine for 3 hours is observed, whereas the total mTOR and p70S6K levels remain relatively constant[1].

In Vivo: Latrepirdine treatment of TgCRND8 transgenic mice is associated with improved learning behavior and with a reduction in accumulation of Aβ42 and α-synuclein. Male, 90-day-old TgCRND8 mice or their wild-type littermates (nTg) receive 31 consecutive once daily i.p. injections of either 3.5 mg/kg Latrepirdine or 0.9% saline (vehicle). At the culmination of treatment, mice are tested for cued and contextual fear conditioning using a paradigm that has been widely accepted for evaluating learning and memory deficits in APP transgenic mice. A significant increase in cued memory only among Latrepirdine-versus vehicle-treated TgCRND8 mice (p=0.01) is observed. A weak, non-significant trend toward an improvement in contextual memory among Latrepirdine-versus vehicle-treated mice (p=0.099) is also observed[1].



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