Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Paroxetine (hydrochloride hemihydrate)

Catalog No: tcsc2873

Available Sizes

Size: 100mg

Size: 500mg

Specifications

CAS No:
110429-35-1

Formula:
$C_{19}H_{20}FNO_3.Cl\ H._1/_2H_2O$

Pathway:
Neuronal Signaling;Autophagy

Target:
Serotonin Transporter;Autophagy

Purity / Grade:
>98%

Solubility:
10 mM in DMSO

Alternative Names:
BRL29060 hydrochloride hemihydrate;BRL29060A hemihydrate

Observed Molecular Weight:
374.83

Product Description

Paroxetine hydrochloride hemihydrate is a potent selective **serotonin-reuptake** inhibitor, commonly prescribed as an antidepressant and has GRK2 inhibitory ability with $\mathbf{IC_{50}}$ of 14 μM.

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IC50 & Target: IC50: 14 μM (GRK2)[3]

In Vitro: Paroxetine (1 μM and 10 μM) distinctly restrains T cell migration induced by CX3CL1 through inhibiting GRK2. Paroxetine inhibits GRK2 induced activation of ERK[1]. Paroxetine (10 μM) reduces pro-inflammatory cytokines in LPS-stimulated BV2 cells. Paroxetine (0-5 μM) leads to a dose-dependent inhibition on LPS-induced production of TNF-α and IL-1β in BV2 cells. Paroxetine also inhibits lipopolysaccharide (LPS)-induced nitric oxide (NO) production and inducible nitric oxide synthase (iNOS) expression in BV2 cells. Paroxetine (5 μM) blocks LPS-induced JNK activation and attenuates baseline ERK1/2 activity in BV2 cells. Paroxetine relieves microglia-mediated neurotoxicity, and suppresses LPS-stimulated pro-inflammatory cytokines and NO in primary microglial cells[4].

In Vivo: Paroxetine treatment obviously attenuates the symptoms of CIA rats. Paroxetine treatment clearly prevents the histological damage of joints and alleviates T cells infiltration into synovial tissue. Paroxetine reveals a strong effect on inhibiting CX3CL1 production in synovial tissues[1]. Paroxetine (20 mg/kg/day) reduces the myocyte cross-sectional area in rat and ROS formation in the remote myocardium. Paroxetine reduces the susceptibility to ventricular tachycardia. Paroxetine treatment following MI decreases LV remodeling and susceptibility to arrhythmias, probably by reducing ROS formation[2]. In CCI paroxetine-treated group, paroxetine (10 mg/kg, i.p.) produces hyperalgesia at days 7 and 10 (P[5].