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Lomerizine dihydrochloride

Catalog No: tcsc0012057

Available Sizes

Size: 50mg

Specifications

CAS No:
101477-54-7

Formula:
$C_{27}H_{32}Cl_2F_2N_2O_3$

Pathway:
Membrane Transporter/Ion Channel

Target:
Calcium Channel

Purity / Grade:
>98%

Solubility:
DMSO : 100 mg/mL (184.69 mM; Need ultrasonic)

Alternative Names:
KB-2796

Observed Molecular Weight:
541.46

Product Description

Lomerizine dihydrochloride is an antagonist of **L-** and **T-type voltagegated calcium channels**.

IC50 & Target: L- and T-type calcium channel[1]

In Vitro: Lomerizine is an antagonist of L- and T-type voltagegated calcium channels and transient receptor potential channel 5

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transient receptor potential channels. Lomerizine is a dual L/T-type channel blocker used for prophylaxis of migraine. To demonstrate the effectiveness of Lomerizine in limiting intracellular [Ca^{2+}], its ability to inhibit glutamate-induced death of motor neurons and the associated rise in cytosolic [Ca^{2+}] is evaluated. Lomerizine inhibits the low- and high-voltage activated Ca^{2+} currents in dissociated rat brain neurons at a threshold concentration of 0.01 μM and IC_{50} of 1.9 μM and H_2O_2-induced Ca^{2+} influx in hippocampal neurons is inhibited by 1 μM Lomerizine. Pre-treatment with 1 μM Lomerizine significantly reduces acute death of motor neurons in spinal cord-DRG cultures exposed to 50 μM glutamate, a concentration that kills approximately 40% of motor neurons in the culture by 6 h, and inhibits the rise in cytosolic [Ca^{2+}] that occurs with glutamate treatment. 0.5 μM Lomerizine is sufficient to significantly prevent the mitochondrial fragmentation of mitochondria induced by SOD1G93A[1]. Lomerizine increases the cytotoxicity of Adriamycin (ADM) and the apoptosis induced by ADM or Vincristine (VCR) in K562/ADM cells. At the concentration of 3, 10 and 30μM, Lomerizine reduces the IC_{50} value of ADM from 79.03 μM to 28.14, 8.16 and 3.16 μM, respectively. Lomerizine increases the intracellular accumulation of ADM and inhibits the efflux of Rh123 in K562/ ADM cells. No change in P-gp expression is observed after the treatment of Lomerizine for 72 h. Lomerizine has strong reversal effect on MDR in K562/ADM cells by inhibiting P-gp function[2].

In Vivo: To determine whether Ca^{2+} signaling molecules mediate NMDA-induced neurotoxicity in p50-deficient mice, the neuroprotective effects of chemical reagents are examined, which act on the Ca^{2+}-signaling pathway including CaN activation, on NMDA-induced RGC death. The p50-deficient mice at 2 months of age, showing normal RGC survival, undergo intraperitoneal pretreatments with a NMDA antagonist, MK801 or Memantine; calcium blocker, Lomerizine; and CaN inhibitor, Tacrolimus, daily for 1 week before the injection of 5 nM NMDA. The chronic administration of Lomerizine or Tacrolimus to KO mice for 6 months results in an increase in surviving RGC numbers (p[3]. Lomerizine (KB-2796; 0.3 and 1 mg/kg, i.v.) dose-dependently increases cerebral blood flow significantly at 30 min and 15 min, respectively, after its administration. Lomerizine (1 mg/kg, i.v.) significantly attenuates the expression of c-Fos-like immunoreactivity in the ipsilateral frontoparietal cortex[4].