

# 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

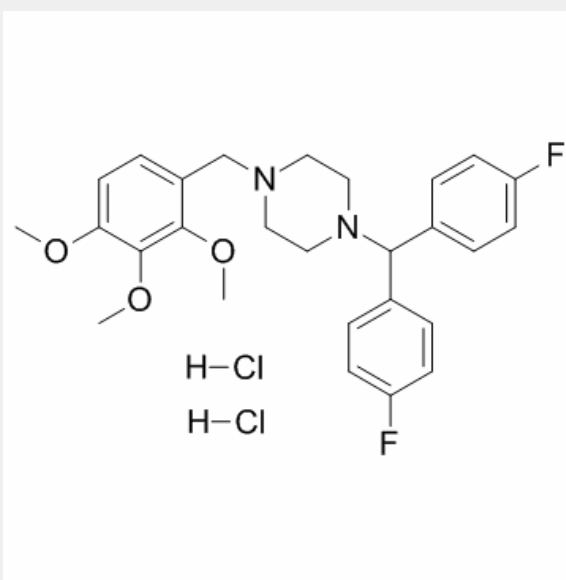
Lomerizine dihydrochloride is an antagonist of **L-** and **T-type voltagegated calcium channels**.

IC50 & Target: L- and T-type calcium channel<sup>[1]</sup>

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

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  $0.01 \mu M$  and  $IC_{50}$  of  $1.9 \mu M$  and  $H_2O_2$ -induced  $Ca^{2+}$  influx in hippocampal neurons is inhibited by  $1 \mu M$  Lomerizine. Pre-treatment with  $1 \mu M$  Lomerizine significantly reduces acute death of motor neurons in spinal cord-DRG cultures exposed to  $50 \mu M$  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.  $0.5 \mu M$  Lomerizine is sufficient to significantly prevent the mitochondrial fragmentation of mitochondria induced by SOD1G93A<sup>[1]</sup>. 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  $30 \mu M$ , Lomerizine reduces the  $IC_{50}$  value of ADM from  $79.03 \mu M$  to 28.14, 8.16 and  $3.16 \mu M$ , 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<sup>[2]</sup>.

**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<sup>[4]</sup>.



All products are for RESEARCH USE ONLY. Not for diagnostic & therapeutic purposes!