

Product Data Sheet

Product Name: Atomoxetine HCl

Cat. No.: GC10274

Chemical Properties

Cas. No. 82248-59-7

Chemical Name (3R)-N-methyl-3-(2-methylphenoxy)-3-phenylpropan-1-amine;hydrochloride

SMILES CC1=CC=CC=C1OC(CCNC)C2=CC=CC=C2.Cl

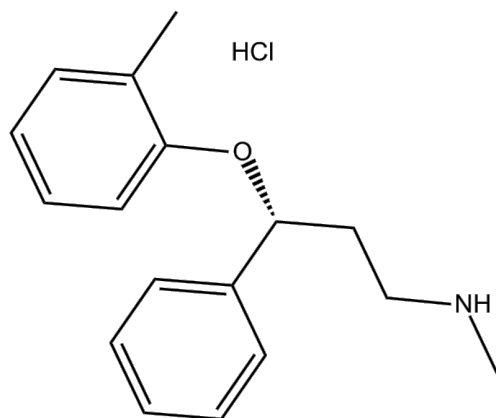
Formula $C_{17}H_{22}ClNO$ M.Wt 291.82

Solubility $\geq 14.35\text{mg/mL}$ in DMSO Storage Store at -20°C

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

Cell lines SH-SY5Y cells

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SH-SY5Y cells were cultured in DMEM/F12 medium supplemented with 10% heat-inactivated fetal bovine serum (FBS) and streptomycin(100µg/ml)/penicillin/(100U/ml) at 37°C in a humidified environment with 5% CO₂. The cells were placed in a medium containing 2% FBS and treated with 10µM retinoic acid (RA), with the medium being replaced every 2 days. The untreated differentiated cells served as the control group. The differentiated cells were treated with 1, 5, 10, 20, and 50µM Atomoxetine HCl for 7 days. After Atomoxetine HCl treatment, the differentiated SH-SY5Y cells were collected and lysed with RIPA lysis buffer containing a mixture of protease and phosphatase inhibitors for immunoblot analysis.

Preparation Method

Reaction Conditions 1, 5, 10, 20, and 50µM; 7 days

Applications Atomoxetine HCl treatment downregulated the mitochondrial fusion markers OPA1 and MFN2 as well as the fission markers DRP1 and Fis1 in the differentiated SH-SY5Y cells.

Animal experiment [2]:

Animal models Male Mongolian gerbils

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Preparation Method	Male Mongolian gerbils (6 months old, weighing 75-80 grams) were raised under pathogen-free (SPF) conditions, with a temperature of 23°C, humidity of 60%, a light/dark cycle of 12 hours/12 hours, and had free access to food and water. Group the gerbils as follows: 1) The sham operation group, which received sham surgery; 2) The ischemia-preconditioned normal saline group, which received normal saline before ischemia-reperfusion; 3) The 30mg/kg Atomoxetine HCl ischemia-preconditioned group, which was given 30mg/kg Atomoxetine HCl before ischemia-reperfusion. Atomoxetine HCl is dissolved in normal saline and is administered intraperitoneally once a day for 3 consecutive days before and after ischemia surgery: the last pre-treatment is performed 30 minutes before the surgery. After ischemia-reperfusion, the brain tissue is collected for analysis.
Dosage form	30mg/kg/day for 3 days; i.p.
Applications	Atomoxetine HCl treatment prevented ischemic neuronal death in the hippocampus of gerbils and significantly reduced the activation of glial cells caused by ischemia.

References:

- [1] Carreón-Trujillo S, Vázquez-González D, Corona J C. Atomoxetine Decreases Mitochondrial Biogenesis, Fission and Fusion In Human Neuron-like Cells But Does Not Alter Antioxidant Defences[J]. Cell Biochemistry and Biophysics, 2023, 81(1): 105-115.
- [2] Park J H, Shin B N, Chen B H, et al. Neuroprotection and reduced gliosis by atomoxetine pretreatment in a gerbil model of transient cerebral ischemia[J]. Journal of the neurological sciences, 2015, 359(1-2): 373-380.

Background

Atomoxetine HCl is a potent and selective norepinephrine uptake inhibitor, with K_i values of 5, 77, and 1451nM for norepinephrine, serotonin (5-HT) and dopamine (DA) [1]. Atomoxetine HCl inhibits cloned human Ether-à-Go-Go-Related Gene (hERG) channels, with an IC_{50} value of 6.3 μ M [2].

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Atomoxetine HCl can regulate the gene expression of neurotransmitter receptors and synaptic signaling molecules in fruit flies, and increase the locomotor activity of wild-type fruit flies^[3]. Atomoxetine HCl has been widely used in animal models of Attention-Deficit/Hyperactivity Disorder (ADHD) to improve hyperactivity symptoms^[4].

In vitro, Atomoxetine HCl treatment (50 μ M) for 7 days induced cell death in human neuron-like cells, resulting in a significant increase in mitochondrial ROS production^[5]. Treatment with 50ng/ml Atomoxetine HCl for 24 hours significantly reduced the expression level of 8-hydroxyguanine glycosylase 1 (hOGG1) in U-937 cells^[6]. In differentiated human SH-SY5Y neuroblastoma cells treated with 50 μ M Atomoxetine HCl for 7 days, the expressions of SDHA and COX-I were significantly downregulated, and mitochondrial fusion and mitochondrial division were disrupted^[7].

In vivo, Atomoxetine HCl treatment for 10 days at a dose of 3mg/kg/day by intraperitoneal, intraperitoneal improved the spatial reference memory of adult rats, and enhanced the attention and inhibitory response control ability of adult rats^[8]. Continuous intraperitoneal injection of Atomoxetine HCl (30mg/kg) once daily for 3 consecutive days can prevent ischemic neuronal death in the hippocampus of gerbils and significantly reduce the activation of glial cells caused by ischemia^[9].

References:

- [1] Bymaster F P, Katner J S, Nelson D L, et al. Atomoxetine increases extracellular levels of norepinephrine and dopamine in prefrontal cortex of rat: a potential mechanism for efficacy in attention deficit/hyperactivity disorder[J]. Neuropsychopharmacology, 2002, 27(5): 699-711.
- [2] Scherer D, Hassel D, Bloehs R, et al. Selective noradrenaline reuptake inhibitor atomoxetine directly blocks hERG currents[J]. British journal of pharmacology, 2009, 156(2): 226-236.
- [3] Qu S, Zhou X, Wang Z, et al. The effects of methylphenidate and atomoxetine on Drosophila brain at single-cell resolution and potential drug repurposing for ADHD treatment[J]. Molecular Psychiatry, 2024, 29(1): 165-185.
- [4] Moon S J, Kim C J, Lee Y J, et al. Effect of atomoxetine on hyperactivity in an animal model of attention-deficit/hyperactivity disorder (ADHD)[J]. PLoS One, 2014, 9(10): e108918.
- [5] Corona J C, Carreón-Trujillo S, González-Pérez R, et al. Atomoxetine produces oxidative stress and alters mitochondrial function in human neuron-like cells[J]. Scientific reports, 2019, 9(1): 13011.
- [6] Schmidt A J, Clement H W, Gebhardt S, et al. Impact of psychostimulants and atomoxetine on the expression of 8-hydroxyguanine glycosylase 1 in human cells[J]. Journal of neural transmission,

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2010, 117(6): 793-797.

[7] Carreón-Trujillo S, Vázquez-González D, Corona J C. Atomoxetine Decreases Mitochondrial Biogenesis, Fission and Fusion In Human Neuron-like Cells But Does Not Alter Antioxidant Defences[J]. Cell Biochemistry and Biophysics, 2023, 81(1): 105-115.

[8] Callahan P M, Plagenhoef M R, Blake D T, et al. Atomoxetine improves memory and other components of executive function in young-adult rats and aged rhesus monkeys[J]. Neuropharmacology, 2019, 155: 65-75.

[9] Park J H, Shin B N, Chen B H, et al. Neuroprotection and reduced gliosis by atomoxetine pretreatment in a gerbil model of transient cerebral ischemia[J]. Journal of the neurological sciences, 2015, 359(1-2): 373-380.

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