

Product Data Sheet

Product Name: Nadolol
Cat. No.: GC39740

Chemical Properties

Cas. No. 42200-33-9

SMILES OC1CC2=C(C(OCC(O)CNC(C)(C)C)=CC=C2)CC1O

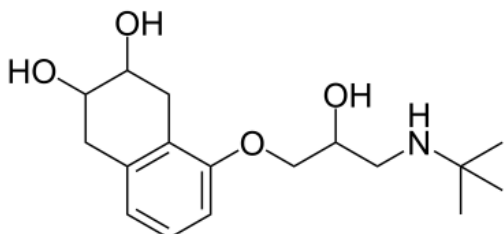
Formula $C_{17}H_{27}NO_4$ M.Wt 309.4

Solubility Methanol: 250 mg/mL (808.02 mM) 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 A549 cells

Caution: Product has not been fully validated for medical applications. For research use only.

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A549 cells were cultured in DMEM/F12 medium supplemented with 10% fetal bovine serum (FBS) and antibiotics (penicillin 100U/ml, streptomycin 0.1mg/ml) under normal conditions (5% CO₂ with 95% humidified air). A549 cells (1×10⁴ Preparation Method per well) were inoculated in a 96-well cell culture plate and incubated at 37°C with 5% CO₂ for 24h. Then, the cells were treated with different concentrations of Nadolol (5, 10, 25, 50, 100, 150, 200, and 250μM) for 48h, respectively, and cell viability was measured.

Reaction
 Conditions 5, 10, 25, 50, 100, 150, 200, and 250μM; 48h

Applications Nadolol treatment reduced the cell viability of A549 cells in a dose-dependent manner.

Animal experiment [2]:

Animal models Male Sprague-Dawley (SD) rats

Male Sprague-Dawley (SD) rats (250-300g) were housed in groups of 3-5 rats per cage with free access to food and water ad libitum. The 12-h light/dark cycles were maintained throughout the duration of animal housing, with light periods beginning consistently at 7:00 am. Before surgery, the rats were fasted overnight but were free to drink tap water. The ischemic stroke rat model was constructed by Middle Cerebral Artery Occlusion (MCAO). Rats were randomly distributed into four groups (n=12 per group): (1) the sham group, (2) the stroke group, (3) the stroke+Nadolol group, rats received Nadolol (5mg/kg; i.p.) at 1h after reperfusion, and (4) the stroke+vehicle group, rats received DMSO (i.p.) at 1h after reperfusion. Following the end of reperfusion, an assessment of the neurological deficit score was first conducted, and then the rats were executed under anesthesia. The brain tissues were preserved for infarct volume measurement.

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Dosage form 5mg/kg for once; i.p.

Applications Nadolol treatment reversed the increases in neurological deficit score and infarct volume in the ischemic stroke rats.

References:

- [1] Kavakcioğlu Yardımcı B, Geyikoglu F, Aysin F, et al. The cytotoxic and apoptotic effects of beta-blockers with different selectivity on cancerous and healthy lung cell lines[J]. Molecular Biology Reports, 2021, 48(5): 4009-4019.
- [2] Yang X Y, Zhu W J, Chen D, et al. Nadolol Attenuates Brain Cell Ferroptosis in Ischemic Stroke Rats by Targeting the HOIL-1/IRP2 Pathway[J]. CNS & Neurological Disorders-Drug Targets, 2025, 24(5): 397-408.

Background

Nadolol is a non-selective and orally active beta-adrenergic antagonist with antihypertensive and antiarrhythmic activities [1]. Nadolol can inhibit the calmodulin-activated Ca^{2+} -ATPase of human erythrocyte membranes [2]. Nadolol has been widely used to reduce heart rate at rest and during exercise and to reduce plasma renin activity[3].

In vitro, Nadolol treatment (10 μM) for 24 hours significantly increased $\text{G}\alpha\text{s}$ protein levels and enhanced basal cAMP levels in $\beta 2\text{AR}$ -expressing HEK 293 cells[4]. Treatment with 250 μM Nadolol for 48h induced apoptosis and decreased mitochondrial membrane potential in A549 cells[5].

In vivo, Nadolol treatment via intraperitoneal injection at a single dose of 20mg/kg significantly reduced the number and area of lung metastases in a B16F10 cell-xenograft mouse model under acute social stress during 15 days[6]. A single dose of 5mg/kg Nadolol was injected intraperitoneally at 1h after reperfusion, which significantly reversed the increases in neurological deficit score and infarct volume in the ischemic stroke rats[7].

References:

- [1] Kalsoom S, Zamir A, Rehman A U, et al. Clinical pharmacokinetics of nadolol: A systematic

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- [2] Meltzer H L, Kassir S. Inhibition of calmodulin-activated Ca^{2+} -ATPase by propranolol and nadolol[J]. Biochimica et Biophysica Acta (BBA)-General Subjects, 1983, 755(3): 452-456.
- [3] Heel R C, Brogden R N, Pakes G E, et al. Nadolol: a review of its pharmacological properties and therapeutic efficacy in hypertension and angina pectoris[J]. Drugs, 1980, 20(1): 1-23.
- [4] Peng H, Bond R A, Knoll B J. The effects of acute and chronic nadolol treatment on β 2AR signaling in HEK293 cells[J]. Naunyn-Schmiedeberg's archives of pharmacology, 2011, 383(2): 209-216.
- [5] Kavakcioğlu Yardımcı B, Geyikoglu F, Aysin F, et al. The cytotoxic and apoptotic effects of beta-blockers with different selectivity on cancerous and healthy lung cell lines[J]. Molecular Biology Reports, 2021, 48(5): 4009-4019.
- [6] Vegas O, Garmendia L, Arregi A, et al. Effects of antalarmin and nadolol on the relationship between social stress and pulmonary metastasis development in male OF1 mice[J]. Behavioural brain research, 2009, 205(1): 200-206.
- [7] Yang X Y, Zhu W J, Chen D, et al. Nadolol Attenuates Brain Cell Ferroptosis in Ischemic Stroke Rats by Targeting the HOIL-1/IRP2 Pathway[J]. CNS & Neurological Disorders-Drug Targets, 2025, 24(5): 397-408.

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