Dilazep dihydrochloride

Cat. No.: HY-100957

CAS No.: 20153-98-4

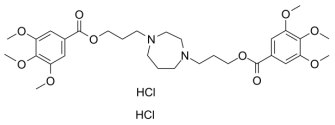
Molecular Formula: $C_{31}H_{46}Cl_2N_2O_{10}$

Molecular Weight: 677.61

Target: Others

Pathway: Others

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (147.58 mM)
DMSO : 66.67 mg/mL (98.39 mM; Need ultrasonic)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	1.4758 mL	7.3789 mL	14.7578 mL
5 mM	0.2952 mL	1.4758 mL	2.9516 mL
10 mM	0.1476 mL	0.7379 mL	1.4758 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (147.58 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (3.69 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (3.69 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (3.69 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Dilazep dihydrochloride is an inhibitor of adenosine uptake. Dilazep dihydrochloride has cerebral and coronary vasodilating action through enhancement of effect of adenosine. Dilazep dihydrochloride also inhibits the ischemic damage, platelet aggregation, and membrane transport of nucleosides[1][2].

IC_{50} & Target

Adenosine uptake

In Vitro	The uptake mechanism has been studied extensively in vitro and Dilazep, NBI and Dipyridamole have been reported to inhibit the uptake of adenosine into different cells. Of these compounds, Dilazep and NBI are almost 10 times more potent than Dipyridamole. In addition, only Dilazep is water soluble and no solubility aiding organic solvent is needed for preparing an aqueous solution[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	After administration of Dilazep, even low doses (0.04-0.1 mg/kg/min) of exogenous adenosine significantly increases superior mesenteric arterial conductance (SMAC) and elevates arterial plasma adenosine concentration. The increased adenosine levels were highly correlated with the increased percentage of change of SMAC and values for R_{max} and EC_{50} were 193.4% change of SMAC and 2.8 μM, respectively. Administration of bolus doses of 8-phenyltheophylline abolishes the ability of Dilazep to potentiate vasodilation, but did not affect isoproterenol-induced relaxation[1]. Dilazep inhibits the phospholipase activation in reperfused heart mitochondria and also inhibits the lipid peroxidation caused by cerebral ischemia and reperfusion. Dilazep may prevent ischemic cerebral injury due to an increase in cerebral blood flow and/or its protective effects on vascular endothelial cell membrane[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Zhang Y, et al. Dilazep potentiation of adenosine-mediated superior mesenteric arterial vasodilation. J Pharmacol Exp Ther. 1991 Sep;258(3):767-71.

[2]. Kawagoe J, et al. Effect of dilazep dihydrochloride against ischemia and reperfusion-induced disruption of blood-brain barrier in rats: a quantitative study. Naunyn Schmiedebergs Arch Pharmacol. 1992 Apr;345(4):485-8.

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