Diazoxide

Cat. No.: HY-B1140

CAS No.: 364-98-7

Molecular Formula: $C_8H_7ClN_2O_2S$

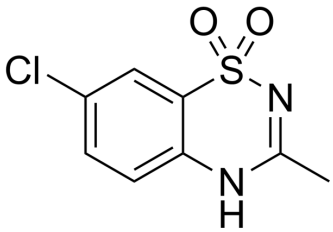
Molecular Weight: 230.67

Target: Potassium Channel; Autophagy

Pathway: Membrane Transporter/Ion Channel; Autophagy

Storage: 4°C, protect from light

* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 35 mg/mL (151.73 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.3352 mL	21.6760 mL	43.3520 mL
5 mM	0.8670 mL	4.3352 mL	8.6704 mL
10 mM	0.4335 mL	2.1676 mL	4.3352 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (9.02 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (9.02 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Diazoxide (Sch-6783) is an ATP-sensitive potassium channel activator, has the potential for hyperinsulinism treatment.

In Vitro

Diazoxide (Sch-6783) has a number of physiological effects, including lowering the blood pressure and rectifying hypoglycemia. Diazoxide has powerful protective properties against cardiac ischemia[1].

Diazoxide (Sch-6783) could protect NSC-34 neurons against the main sources of neurodegenerative damage. Diazoxide increases Nrf2 nuclear translocation in NSC-34 motoneurons and prevents endogenous oxidative damage[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Diazoxide (Sch-6783) attenuates postresuscitation brain injury, protects mitochondrial function, inhibits brain cell apoptosis, and activates the PKC pathway by opening mitoKATP channels[3].

Treatment with Diazoxide (Sch-6783) in wild-type mice decreases intraocular pressure (IOP) by 21.5±3.2% with an absolute IOP reduction of 3.9 ± 0.6 mm Hg[4].

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PROTOCOL

Cell Assay [2]

Diazoxide is dissolved in DMSO to prepare 50 mM stock solution. NSC-34 cells are allowed to differentiate for 8 weeks under reduced serum conditions and then seeded in 24-well plates. Glutamate is dissolved in culture medium and added to cultures at concentration of 10 μM for 24 h. Cell treatment with 100 μM diazoxide starts 2 h before glutamate exposure. Cell viability is measured by the MTT assay[2].

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Animal Administration [3][4]

Rats: Adult male Sprague-Dawley rats with induced cerebral ischemia (n=10 per group) receive an intraperitoneal injection of 0.1% DMSO (1 mL; vehicle group), diazoxide (10 mg/kg; DZ group), or diazoxide (10 mg/kg) plus 5-hydroxydecanoate (5 mg/kg; DZ + 5-HD group) 30 min after CPR. The control group (sham group, n=5) undergoes sham operation, without cardiac arrest. Mitochondrial respiratory control rate (RCR) is determined. Brain cell apoptosis is assessed using TUNEL staining. Expression of Bcl-2, Bax, and protein kinase C epsilon (PKCε) in the cerebral cortex is determined by Western blotting and immunohistochemistry[3].

Mouse: Diazoxide is prepared by diluting a 100 mM stock solution in 10% polyethoxylated castor oil in PBS. In C57BL/6 wild-type and Kir6.2$^{(-/-)}$ mice, a 5 μL drop of 5 mM diazoxide is topically administered to one eye of each mouse while the fellow control eye received vehicle (DMSO and 10% polyethoxylated castor oil in the same proportion as the treated eye). IOP is measured daily at 1 hour, 4 hours, and 23 hours following treatment. Treatment with diazoxide and vehicle is continued daily for 14 consecutive days[4].

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CUSTOMER VALIDATION

- Redox Biol. 15 October 2021, 102168.
- Cell Biol Int. 2020 Jun;44(6):1353-1362.
- Biological Sciences. 2020 Sep.

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REFERENCES

[1]. Coetzee WA, et al. Multiplicity of effectors of the cardioprotective agent, diazoxide. Pharmacol Ther. 2013 Nov;140(2):167-75.

[2]. Virgili N, et al. K(ATP) channel opener diazoxide prevents neurodegeneration: a new mechanism of action viaantioxidative pathway activation. PLoS One. 2013 Sep 11;8(9):e75189.

[3]. Wu H, et al. Diazoxide Attenuates Postresuscitation Brain Injury in a Rat Model of Asphyxial Cardiac Arrest by Opening Mitochondrial ATP-Sensitive Potassium Channels. Biomed Res Int. 2016;2016:1253842.

[4]. Chowdhury UR, et al. ATP-sensitive potassium (K(ATP)) channel openers diazoxide and nicorandil lower intraocular pressure in vivo. Invest Ophthalmol Vis Sci. 2013 Jul 22;54(7):4892-9.

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

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