Glibenclamide

Cat. No.: HY-15206

CAS No.: 10238-21-8

Molecular Formula: $C_{23}H_{28}ClN_3O_5S$

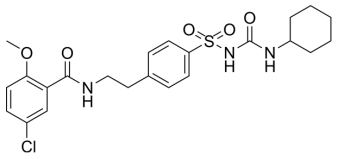
Molecular Weight: 494

Target: Potassium Channel; Autophagy; CFTR; P-glycoprotein; Mitochondrial Metabolism

Pathway: Membrane Transporter/Ion Channel; Autophagy; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (202.43 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.0243 mL	10.1215 mL	20.2429 mL
5 mM	0.4049 mL	2.0243 mL	4.0486 mL
10 mM	0.2024 mL	1.0121 mL	2.0243 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: ≥ 2.5 mg/mL (5.06 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.06 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Glibenclamide (Glyburide) is an orally active ATP-sensitive K^+ channel (K_{ATP}) inhibitor and can be used for the research of diabetes and obesity[1]. Glibenclamide inhibits P-glycoprotein. Glibenclamide directly binds and blocks the SUR1 subunits of K_{ATP} and inhibits the cystic fibrosis transmembrane conductance regulator protein (CFTR)[3]. Glibenclamide interferes with mitochondrial bioenergetics by inducing changes on membrane ion permeability[4]. Glibenclamide can induce autophagy[5].

IC_{50} & Target

K_{ATP}[1]

In Vitro

Glibenclamide (Brown adipocytes; 10 μM; 1 day) has no effect on adipocyte differentiation. Glibenclamide (Ucp1-2A-GFP

brown adipocyte) significantly increases UCP1 expression. Glibenclamide directly binds and blocks the SUR1 subunits of ATP-dependent potassium channels (K_{ATP}) and consequently increases insulin secretion from the pancreatic β cells[2]. Glibenclamide interferes with mitochondrial bioenergy by permeating mitochondrial intima with Cl^- and promoting mitochondrial net Cl^-/K^+ cotransport[4]. Glibenclamide induced autophagy inhibits its insulin secretion-improving function in β cells[5].

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

In Vivo

Glibenclamide (2 mg/kg; p.o.) increases of insulin release and rapid drop of blood glucose level[2].

Glibenclamide (50 μg/kg; p.o.) does not cause significant change, such as body weight or body composition[2].

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

Animal Model:	Mice[2]
Dosage:	2 mg/kg
Administration:	P.o.
Result:	Increased of insulin release and rapid drop of blood glucose level.

CUSTOMER VALIDATION

- Nat Metab. 2022 Feb;4(2):269-283.
- Pharmacol Res. 2020 Mar;153:104679.
- Br J Pharmacol. 2020 May;177(10):2286-2302.
- J Cell Physiol. 2023 Sep 8.
- Front Pharmacol. 26 April 2021.

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REFERENCES

[1]. Fernandes MA, et al. Glibenclamide interferes with mitochondrial bioenergetics by inducing changes on membrane ion permeability. J Biochem Mol Toxicol. 2004;18(3):162-169.

[2]. Heo R, et al. The anti-diabetic drug trelagliptin induces vasodilation via activation of Kv channels and SERCA pumps. Life Sci. 2021;283:119868.

[3]. Qiu Y, et al. Glyburide Regulates UCP1 Expression in Adipocytes Independent of KATP Channel Blockade. iScience. 2020;23(9):101446.

[4]. Golstein PE, et al. P-glycoprotein inhibition by glibenclamide and related compounds. Pflugers Arch. 1999;437(5):652-660.

[5]. Zhou J, et al. Glibenclamide-Induced Autophagy Inhibits Its Insulin Secretion-Improving Function in β Cells. Int J Endocrinol. 2019;2019:1265175.

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

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