Metformin hydrochloride

Cat. No.: HY-17471A

CAS No.: 1115-70-4

Molecular Formula: $C_4H_{12}ClN_5$

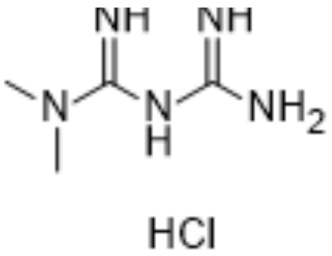
Molecular Weight: 165.62

Target: AMPK; Autophagy; Mitophagy; Apoptosis; mTOR

Pathway: Epigenetics; PI3K/Akt/mTOR; Autophagy; Apoptosis

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 (603.79 mM)

DMSO : ≥ 1.7 mg/mL (10.26 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	6.0379 mL	30.1896 mL	60.3792 mL
	5 mM	1.2076 mL	6.0379 mL	12.0758 mL
	10 mM	0.6038 mL	3.0190 mL	6.0379 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 (603.79 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 5% DMSO >> 40% PEG300 >> 5% Tween-80 >> 50% saline
   Solubility: ≥ 3 mg/mL (18.11 mM); Clear solution
3. Add each solvent one by one: 5% DMSO >> 95% (20% SBE-β-CD in saline)
   Solubility: ≥ 3 mg/mL (18.11 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Metformin (1,1-Dimethylbiguanide) hydrochloride inhibits the mitochondrial respiratory chain in the liver, leading to AMPK activation and enhancing insulin sensitivity, and can be used in the study of type 2 diabetes. Metformin hydrochloride also inhibits liver oxidative stress, nitrosative stress, inflammation, and apoptosis caused by liver ischemia/reperfusion injury. In addition, metformin hydrochloride regulates the expression of autophagy-related proteins by activating AMPK and inhibiting the mTOR signaling pathway, thereby inducing tumor cell autophagy and inhibiting the growth of renal cell carcinoma in vitro and in vivo[1][2][3][4][5][6][7].

IC_{50} & Target	AMPK
In Vitro	Metformin hydrochloride (1,1-Dimethylbiguanide hydrochloride) inhibits proliferation of ESCs in a concentration-dependent manner. The IC_{50} is 2.45?mM for A-ESCs and 7.87?mM for N-ESCs. Metformin shows pronounced effects on activation of AMPK signaling in A-ESCs from secretory phase than in cells from proliferative phase[2]. Metformin hydrochloride (0-500 μM) decreases glycogen synthesis in a dose-dependent manner with an IC_{50} value of 196.5 μM in cultured rat hepatocytes[3]. Metformin hydrochloride shows cell viability and cytotoxic effects on PC-3 cells with IC_{50} of 5 mM[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Metformin hydrochloride (1,1-Dimethylbiguanide hydrochloride; 100 mg/kg, p.o.) alone, and metformin (25, 50, 100 mg/kg) with NSC 37745 groups attenuates myocyte necrosis through histopathological analysis[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	ESCs are plated in 96-well plates at a concentration of 1×10^3cells/well. After attachment, cells are treated with different doses of metformin/compound C for 0 min, 15 min, 1 h, and 24 h. MTT assays are performed as described previously. In brief, MTT (5 mg/mL) is added to the 96-well plates at a volume of 10 μL/well, and the plates are incubated for 4 h. The MTT reaction is terminated by removal of the culture medium containing MTT, and 100 μL DMSO per well are added and incubated at RT on a shaker for 10 min to ensure that the crystals had dissolved sufficiently. Absorbance values are measured at 595 nm. Cell proliferation (percentage of control) is calculated as follows: absorbance (experimental group)/absorbance (control group). Cell proliferation inhibition (percentage of control) is calculated as follows: 100%−cell proliferation (percentage of control). Each experiment is performed in duplicate and repeated six times to assess result consistency. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	The animals are randomized into six groups consisting of six rats each. Rats in group 1 (control) receives a subcutaneous injection of physiological saline (0.5 mL) and are left untreated for the entire experimental period. Rats in group 2 receives an oral administration of metformin (100 mg/kg; twice daily) for 2 days and are subcutaneously injected with saline at an interval of 24 h for 2 consecutive days. Rats in group 3 (MI control) receives an oral administration of saline (twice daily) for 2 days and are sc injected with NSC 37745 (100 mg/kg) daily for 2 consecutive days at an interval of 24 h. Rats in groups 4 to 6 are treated with metformin at 25, 50, and 100 mg/kg. Metformin is dissolved in saline and is gavaged at a volume of 0.25-0.5 mL twice a day at an interval of 12 h, started immediately before NSC 37745 injection. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nature. 2023 Sep;621(7977):188-195.
- Cancer Cell. 2020 Sep 14;38(3):350-365.e7.
- Cell Res. 2023 Jul 17.
- Signal Transduct Target Ther. 2023 Mar 6;8(1):95.
- Signal Transduct Target Ther. 2020 May 20;5(1):56.

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REFERENCES

[1]. Quaile MP, et al. Toxicity and toxicokinetics of metformin in rats. Toxicol Appl Pharmacol. 2010 Mar 15;243(3):340-7.

[2]. Abdel-Zaher AO, et al. Novel mechanistic insights of the potential role of gasotransmitters and autophagy in the protective effect of metformin against hepatic ischemia/reperfusion injury in rats. Naunyn Schmiedebergs Arch Pharmacol. 2025 Feb 6.

[3]. Liu J, et al. Metformin inhibits renal cell carcinoma in vitro and in vivo xenograft. Urol Oncol. 2013 Feb;31(2):264-70.

[4]. Soraya H, et al. Acute treatment with metformin improves cardiac function following NSC 37745 induced myocardial infarction in rats. Pharmacol Rep. 2012;64(6):1476-84.

[5]. Xue J, et al. Metformin inhibits growth of eutopic stromal cells from adenomyotic endometrium via AMPK activation and subsequent inhibition of AKT phosphorylation: a possible role in the treatment of adenomyosis. Reproduction. 2013 Aug 21;146(4):397-406.

[6]. Otto M, et al. Metformin inhibits glycogen synthesis and gluconeogenesis in cultured rat hepatocytes. Diabetes Obes Metab. 2003 May;5(3):189-94.

[7]. Avci CB, et al. Therapeutic potential of an anti-diabetic drug, metformin: alteration of miRNA expression in prostate cancer cells. Asian Pac J Cancer Prev. 2013;14(2):765-8.

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