Metformin

Cat. No.: HY-B0627

CAS No.: 657-24-9

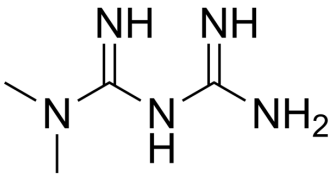
Molecular Formula: $C_4H_{11}N_5$

Molecular Weight: 129.17

Target: AMPK; Autophagy; Mitophagy; Apoptosis; mTOR

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

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : 50 mg/mL (387.10 mM; Need ultrasonic)
DMSO : 25 mg/mL (193.55 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	7.7419 mL	38.7096 mL	77.4192 mL
5 mM	1.5484 mL	7.7419 mL	15.4838 mL
10 mM	0.7742 mL	3.8710 mL	7.7419 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 (774.19 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.08 mg/mL (16.10 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.08 mg/mL (16.10 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Metformin (1,1-Dimethylbiguanide) 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 also inhibits liver oxidative stress, nitrosative stress, inflammation, and apoptosis caused by liver ischemia/reperfusion injury. In addition, metformin 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 (1,1-Dimethylbiguanide) 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[3].
Metformin (0-500 μM) decreases glycogen synthesis in a dose-dependent manner with an IC_{50} value of 196.5 μM in cultured rat hepatocytes[4].
Metformin shows cell viability and cytotoxic effects on PC-3 cells with IC_{50} of 5 mM[5].
Metformin (1–50 mM; 0-120 h) significantly inhibites the proliferation of both 786-O and OS-RC-2 RCC cell lines in a dose- and time-dependent manner[7].
Metformin (5 mM; 0-48 h) stimulates AMPK and inhibited the mTOR signaling pathway in 786-O cells[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[7]

Cell Line:	786-O and OS-RC-2 cells
Concentration:	1□5□10□20 and 50 mM
Incubation Time:	0-120 h
Result:	Inhibited the growth of 786-O and OS-RC-2 cells by 10% and 14% at doses of 0.2 and 0.5 mM, respectively.

Western Blot Analysis[7]

Cell Line:	786-O cells
Concentration:	5 mM
Incubation Time:	0-48 h
Result:	Reduced phosphorylation of S6K1 (Thr389) in a time-dependent manner, and thus reduced phosphorylation of ribosomal S6 protein (Ser235/236). Activated AMPK in a time-dependent manner.

In Vivo

Metformin (1,1-Dimethylbiguanide; 100 mg/kg, p.o.) alone, and metformin (25, 50, 100 mg/kg) with isoproterenol groups attenuates myocyte necrosis through histopathological analysis[1].
Metformin (> 900 mg/kg/day, p.o.) results in moribundity/mortality and clinical signs of toxicity in Crl:CD(SD) rats[2].
Metformin (200 mg/kg; i.p.; once a day for 6 consecutive days before induction of ischemia and at the beginning of reperfusion) significantly reduces the serum alanine aminotransferase (ALT) level in rats with ischemia-reperfusion model[6].
Metformin (250 mg/kg; i.p.; once daily for 22 days) significantly reduces the growth of 786-O cell xenograft tumors in nude mice[7].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nature. 2025 Feb;638(8052):1112-1121.
- 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.

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REFERENCES

[1]. 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.

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

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

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

[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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