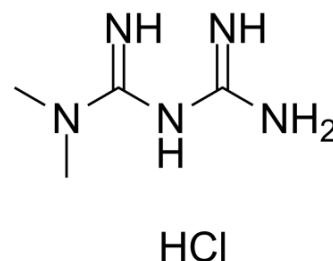


Metformin hydrochloride

Cat. No.:	HY-17471A
CAS No.:	1115-70-4
Molecular Formula:	C ₄ H ₁₂ ClN ₅
Molecular Weight:	165.62
Target:	AMPK; Autophagy; Mitophagy
Pathway:	Epigenetics; PI3K/Akt/mTOR; Autophagy
Storage:	4°C, protect from light
	* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

H₂O : ≥ 32 mg/mL (193.21 mM)
 DMSO : ≥ 1.7 mg/mL (10.26 mM)
 * "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Solvent	Mass	1 mg	5 mg	10 mg
	Concentration				
	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.

BIOLOGICAL ACTIVITY

Description

Metformin hydrochloride (1,1-Dimethylbiguanide hydrochloride) inhibits the **mitochondrial respiratory chain** in the liver, leading to activation of **AMPK**, enhancing insulin sensitivity for type 2 diabetes research. Metformin hydrochloride triggers **autophagy**^[1].

IC₅₀ & Target

AMPK

In Vitro

Metformin hydrochloride (1,1-Dimethylbiguanide hydrochloride) inhibits proliferation of ESCs in a concentration-dependent manner. The IC₅₀ 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 IC₅₀ value of 196.5 μM in cultured rat hepatocytes^[3]. Metformin hydrochloride shows cell viability and cytotoxic effects on PC-3 cells with IC₅₀ 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 1×10^3 cells/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 μ L/well, and the plates are incubated for 4 h. The MTT reaction is terminated by removal of the culture medium containing MTT, and 100 μ L 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

- **Adv Mater.** 2020 Oct 5;e2003708.
- **Cancer Cell.** 2020 Sep 14;38(3):350-365.e7.
- **Signal Transduct Target Ther.** 2020 May 20;5(1):56.
- **Nat Commun.** 2019 Feb 6;10(1):620.
- **Cell Rep.** 2019 Oct.

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REFERENCES

[1]. 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.

[2]. 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.

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

[4]. 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.

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

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