Phenformin hydrochloride

Cat. No.: HY-16397A

CAS No.: 834-28-6

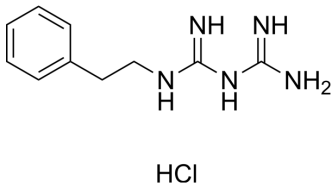
Molecular Formula: $C_{10}H_{16}ClN_5$

Molecular Weight: 241.72

Target: AMPK; Autophagy

Pathway: Epigenetics; PI3K/Akt/mTOR; Autophagy

Storage: 4°C, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 12.5 mg/mL (51.71 mM; Need ultrasonic)
DMSO : 10 mg/mL (41.37 mM; Need ultrasonic)

Preparing Stock Solutions

Concentration / Solvent / Mass	1 mg	5 mg	10 mg
1 mM	4.1370 mL	20.6851 mL	41.3702 mL
5 mM	0.8274 mL	4.1370 mL	8.2740 mL
10 mM	0.4137 mL	2.0685 mL	4.1370 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 2.5 mg/mL (10.34 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.42 mg/mL (1.74 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.42 mg/mL (1.74 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.42 mg/mL (1.74 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Phenformin hydrochloride is an anti-diabetic agent from the biguanide class, can activate AMPK activity.

IC_{50} & Target AMPK

In Vitro Phenformin stimulates the phosphorylation and activation of AMPKalpha1 and AMPKalpha2 without altering LKB1 activity[1].

	Phenformin increases AMPK activity and phosphorylation in the isolated heart, the increase in AMPK activity is always preceded by and correlated with increased cytosolic [AMP][2].? Phenformin is a 50-fold more potent inhibitor of mitochondrial complex I than metformin. Phenformin robustly induces apoptosis in LKB1 deficient NSCLC cell lines. Phenformin at 2 mM similarly induces AMPK signaling as shown by increased P-AMPK and P-Raptor levels. Phenformin induces higher levels of cellular stress, triggering induction of P-Ser51 eIF2α and its downstream target CHOP, and markers of apoptosis at later times. Phenformin induces a significant increase in survival and therapeutic response in KLluc mice following long-term treatment[3].? Phenformin and AICAR increases AMPK activity in H441 cells in a dose-dependent fashion, stimulating the kinase maximally at 5-10 mm and 2 mm, respectively. Phenformin significantly decreases basal ion transport (measured as short circuit current) across H441 monolayers by approximately 50% compared with that of controls. Phenformin and AICAR significantly reduce amiloride-sensitive transepithelial Na+ transport compared with controls. Phenformin and AICAR suppress amiloride-sensitive Na^+ transport across H441 cells via a pathway that includes activation of AMPK and inhibition of both apical Na+ entry through ENaC and basolateral Na^+ extrusion via the Na^+,K^+-ATPase[4].? Phenformin-treated rats reveals a tendency towards a decrease in blood insulin level (radioimmunoassay)[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Phenformin increases levels of P-eIF2α and its target BiP/Grp78 in normal lung as well as in lung tumors of mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Kinase Assay [2]	Total AMPK activity is measured using the method of Dagher et al. AMPK activity is quantified in the resuspended pellet as incorporation of ^{32}P from [γ-^{32}P]ATP (10 GBq/mmol) into a synthetic peptide with the specific target sequence for AMPK, the SAMS peptide. Radioactivity is measured using a liquid scintillation counter. Protein content in the solution containing the resupended $(NH4)_2SO_4$ pellet is determined using the Bradford method. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Nat Commun. 2019 Jul 1;10(1):2901.
- Cell Rep Med. 2022 Nov 3;100802.
- Mol Syst Biol. 2023 Jun 1;e11267.
- Biomed Pharmacother. 2020 Aug;128:110216.
- Life Sci. 2019 Jan 15;217:243-250.

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REFERENCES

[1]. Sakamoto K, et al. Activity of LKB1 and AMPK-related kinases in skeletal muscle: effects of contraction, phenformin, and AICAR. Am J Physiol Endocrinol Metab.?2004 Aug;287(2):E310-7.

[2]. Zhang L, et al. Metformin and phenformin activate AMP-activated protein kinase in the heart by increasing cytosolic AMP concentration. Am J Physiol Heart Circ Physiol. 2007 Jul;293(1):H457-66.

[3]. Moreira AL, et al. Thalidomide exerts its inhibitory action on tumor necrosis factor alpha by enhancing mRNA degradation. J Exp Med. 1993 Jun 1;177(6):1675-80.

[4]. Woollhead AM, et al. Phenformin and 5-aminoimidazole-4-carboxamide-1-beta-D-ribofuranoside (AICAR) activation of AMP-activated protein kinase inhibits transepithelial Na+ transport across H441 lung cells. J Physiol. 2005 Aug 1;566(Pt 3):781-92. Epub 2005

[5]. Dilman VM, et al. Inhibition of DMBA-induced carcinogenesis by phenformin in the mammary gland of rats. Arch Geschwulstforsch. 1978;48(1):1-8.

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