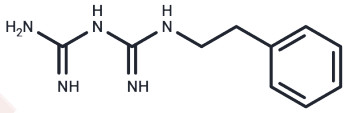
Data Sheet (Cat.No.T20158)

TargetMol

Phenformin

Chemical Properties



CAS No. : 114-86-3

Formula: C10H15N5

Molecular Weight: 205.26

Storage: Keep away from direct sunlight,Store at low temperature,Keep away from moisture,Store under nitrogen
Powder: -20°C for 3 years | In solvent: -80°C for 1 year
Actual storage temperature shall be subject to the COA.

Biological Description

Description	Phenformin (BRN 1977317) is a biguanide compound with anticancer and anti-glycemic activity, inhibits skin tumor growth and promotes keratinocyte differentiation and apoptosis.
Targets(IC50)	Apoptosis,Mitochondrial Metabolism,AMPK
In vitro	Phenformin acts by promoting phosphorylation and activation of AMPKalpha1 and AMPKalpha2 without altering the activity of the LKB1 gene [1]. In isolated hearts, Phenformin increased AMPK activity and phosphorylation levels, which correlated with increased AMPK activity and increased intracellular [AMP] [2].Phenformin was 50 times more potent than Metformin and was able to inhibit mitochondrial complex I. In LKB1-deficient non-small cell lung cancer cell lines, the Phenformin was able to induce apoptosis.Phenformin (2 mM) also induced AMPK signaling and increased levels of P-AMPK and P-Raptor.Phenformin induced higher levels of cellular stress, which in turn triggered the induction of P-Ser51 eIF2α and its downstream target CHOP, which are late stage molecular markers of apoptosis. In chronically treated KLluc mice, Phenformin induced survival and treatment response [3]. In H441 cells, Phenformin and AICAR increased AMPK activity in a dose-dependent manner, maximizing activation of the kinase at 5-10 mM and 2 mM concentrations. In H441 cell monolayers, Phenformin significantly reduced basal ion migration (measured as short-circuit current) by approximately 50%. Phenformin and AICAR significantly reduced amiloride-sensitive transmembrane Na ion transport compared to controls. Phenformin and AICAR inhibited amiloride-sensitive transmembrane Na ion transport in H441 cells by activating AMPK and inducing inhibition of sodium ion entry at the terminal and basolateral sides of ENaC cells via the Na+, K+ ATPase [4]. In rats treated with Phenformin, their blood insulin levels tend to decrease (radioimmunoassay) [5].

Solubility Information

Solubility	DMSO: Soluble, (< 1 mg/ml refers to the product slightly soluble or insoluble)

Preparing Stock Solutions

	1mg	5mg	10mg
1 mM	4.8719 mL	24.3593 mL	48.7187 mL
5 mM	0.9744 mL	4.8719 mL	9.7437 mL
10 mM	0.4872 mL	2.4359 mL	4.8719 mL
50 mM	0.0974 mL	0.4872 mL	0.9744 mL

Please select the appropriate solvent to prepare the stock solution, according to the solubility of the product in different solvents. Please use it as soon as possible.

Note: The dilution table applies only to solid products. For liquid products, please calculate the stock solution based on the stated concentration and/or density.

Reference

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.
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.
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.
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.
Dilman VM, et al. Inhibition of DMBA-induced carcinogenesis by phenformin in the mammary gland of rats. Arch Geschwulstforsch. 1978;48(1):1-8. PMID: 418748.