Folic acid

Cat. No.: HY-16637

CAS No.: 59-30-3

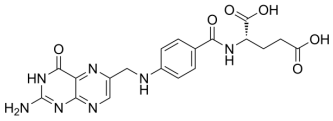
Molecular Formula: $C_{19}H_{19}N_7O_6$

Molecular Weight: 441.4

Target: DNA/RNA Synthesis; Endogenous Metabolite

Pathway: Cell Cycle/DNA Damage; Metabolic Enzyme/Protease

Storage: 4°C, protect from light
* In solvent : -80°C, 2 years; -20°C, 1 year (protect from light)



SOLVENT & SOLUBILITY

In Vitro

1M NaOH : 100 mg/mL (226.55 mM; Need ultrasonic)
DMSO : 33.33 mg/mL (75.51 mM; Need ultrasonic)
H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.2655 mL	11.3276 mL	22.6552 mL
5 mM	0.4531 mL	2.2655 mL	4.5310 mL
10 mM	0.2266 mL	1.1328 mL	2.2655 mL

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

In Vivo

1. Add each solvent one by one: 300 mM NaHCO3 in water
   Solubility: 50 mg/mL (113.28 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 (4.71 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.08 mg/mL (4.71 mM); Suspended solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Folic acid (Vitamin B9) is a orally active essential nutrient from the B complex group of vitamins. Folic acid shows antidepressant-like effect. Folic acid sodium reduces the risk of neonatal neural tube defects. Folic acid can be used to the research of megaloblastic and macrocytic anemias due to folic deficiency[1][2][3][4].

IC_{50} & Target		
	Human Endogenous Metabolite	Microbial Metabolite

In Vitro

Folic acid plays a critical role in the prevention of chromosome breakage and hypomethylation of DNA[1].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Folic acid (10, 50, 100 mg/kg; p.o.) shows an antidepressant-like effect in this behavioral mouse model[2].

Folic acid (1, 10 nmol/site) shows no psychostimulant effect in mice habituated to the novel environment[2].

Folic acid (1, 5 mg/kg; p.o.) prevents epigenetic modification of hepatic gene expression in the offspring in rats[3]

.When folic acid was administrated orally as aqueous solution in rat, the AUC was 1.4 μg h/mL and oral bioavailability is 35%[5].

Induction of Acute Kidney Injury (AKI)[6]

- Background

  Folic acid metabolism requires higher levels of NADPH to reduce folate to THF decreasing the antioxidant defense. The redox imbalance generated by folic acid metabolism is one of the main mechanisms involved in renal damage.

- Specific Mmodeling Methods

  Rat: Wistar • male

  Administration: 300mg/ml • i.p. • single dose

  Note

  (1) Intraperitoneal injection 300 mg/kg folic acid (dissolved in 300 mM $NaHCO_3$) in male Wistar rats with an initial body weight between 230 to 250 g.

  (2) Plasma was collected and analyzed at days 2, 4, 7, 14 and 28 after folic acid administration.

- Modeling Indicators

  Metabolic changes : Assessment of renal injury by blood urea nitrogen (BUN) and plasma creatinine.

  Individual phenotypic change: The ratio of kidney weight to total rat weight was detected.

- Correlated Product(s): Acetylcysteine (HY-B0215)
- Opposite Product(s):

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	30-40 g swiss mice[2]
Dosage:	10, 50, 100 mg/kg

Administration:	Oral administration
Result:	Decreased the immobility time in the forced swimming test (FST) (F_{324}=11.21) and produced a significant effect in the immobility time in the tail suspension test (TST) ($F_{3,20}$ =5.71).

Animal Model:	30-40 g swiss mice[2]
Dosage:	1-10 nmol/site
Administration:	Intracerebroventrical injection
Result:	Decreased the immobility time of mice in the FST ($F_{3,22}$=12.31) and TST ($F_{3,22}$=5.50).

Animal Model:	Virgin female Wistar rats[3]
Dosage:	1, 5 mg/kg (180 g/kg protein with 1 mg/kg folic acid or 90 g/kg casein with 1, 5 mg/kg folic acid)
Administration:	Oral administration
Result:	Prevented epigenetic modification of hepatic gene expression in the offspring.

CUSTOMER VALIDATION

- Cell Rep Med. 2023 Feb 14;100953.
- Br J Cancer. 2023 Jun 27.
- JCI Insight. 2022 Mar 1;e152330.
- J Mol Med (Berl). 2019 Aug;97(8):1183-1193.
- Microbiol Spectr. 2023 Sep 21;e0267123.

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REFERENCES

[1]. Butterworth CE Jr, et al. Folic acid safety and toxicity: a brief review. Am J Clin Nutr. 1989 Aug;50(2):353-8.

[2]. Brocardo PS, et al. Folic acid administration produces an antidepressant-like effect in mice: evidence for the involvement of the serotonergic and noradrenergic systems. Neuropharmacology. 2008 Feb;54(2):464-73.

[3]. Lillycrop KA, et al. Dietary protein restriction of pregnant rats induces and folic acid supplementation prevents epigenetic modification of hepatic gene expression in the offspring. J Nutr. 2005 Jun;135(6):1382-6.

[4]. Pietrzik K, et al. Folic acid and L-5-methyltetrahydrofolate: comparison of clinical pharmacokinetics and pharmacodynamics. Clin Pharmacokinet. 2010 Aug;49(8):535-48.

[5]. Peñalva R, et al. Zein nanoparticles for oral folic acid delivery[J]. Journal of Drug Delivery Science and Technology, 2015, 30: 450-457.

[6]. Aparicio-Trejo OE, et al. Chronic impairment of mitochondrial bioenergetics and β-oxidation promotes experimental AKI-to-CKD transition induced by folic acid. Free Radic Biol Med. 2020 Jul;154:18-32.

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

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