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Proteins

Product Data Sheet



Folic acid-13C₆

Cat. No.: HY-16637S5 CAS No.: 161406-20-8 Molecular Formula: $C_{13}^{13}C_6H_{19}N_7O_6$

447.35 Molecular Weight:

Target: DNA/RNA Synthesis; Endogenous Metabolite; Isotope-Labeled Compounds

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

Please store the product under the recommended conditions in the Certificate of Storage:

Analysis.

BIOLOGICAL ACTIVITY

Description

Folic acid-13C6 is a deuterated labeled Folic $acid^{[1]}$. 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 [2][3][4][5]

In Vitro

Stable heavy isotopes of hydrogen, carbon, and other elements have been incorporated into drug molecules, largely as tracers for quantitation during the drug development process. Deuteration has gained attention because of its potential to affect the pharmacokinetic and metabolic profiles of drugs[1].

Folic acid plays a critical role in the prevention of chromosome breakage and hypomethylation of DNA^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Folic acid plays a critical role in the prevention of chromosome breakage and hypomethylation of DNA^[2].

Folic acid (10, 50, 100 mg/kg; p.o.) shows an antidepressant-like effect in this behavioral mouse model^[3]. Folic acid (1, 10 nmol/site) shows no psychostimulant effect in mice habituated to the novel environment^[3]. Folic acid (1, 5 mg/kg; p.o.) prevents epigenetic modification of hepatic gene expression in the offspring in rats^[4]. When folic acid was administrated orally as aqueous solution in rat, the AUC was 1.4 µg h/mL and oral bioavailability is 35% [6].

Induction of Acute Kidney Injury (AKI)^[7]

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 NaHCO₃) 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)

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

REFERENCES

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- [4]. 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.
- [5]. 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.
- [6]. Pietrzik K, et al. Folic acid and L-5-methyltetrahydrofolate: comparison of clinical pharmacokinetics and pharmacodynamics. Clin Pharmacokinet. 2010 Aug;49(8):535-48.
- [7]. Russak EM, et al. Impact of Deuterium Substitution on the Pharmacokinetics of Pharmaceuticals. Ann Pharmacother. 2019 Feb;53(2):211-216.

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

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