D-Pantothenic acid sodium

Cat. No.: HY-B0430A

CAS No.: 867-81-2

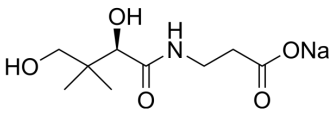
Molecular Formula: $C_9H_{16}NNaO_5$

Molecular Weight: 241.22

Target: Apoptosis; Endogenous Metabolite

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage: 4°C, sealed storage, away from moisture
* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (414.56 mM; Need ultrasonic)
DMSO : 5 mg/mL (20.73 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	4.1456 mL	20.7280 mL	41.4559 mL
5 mM	0.8291 mL	4.1456 mL	8.2912 mL
10 mM	0.4146 mL	2.0728 mL	4.1456 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 120 mg/mL (497.47 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 0.5 mg/mL (2.07 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 0.5 mg/mL (2.07 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 0.5 mg/mL (2.07 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

D-Pantothenic acid sodium (Sodium pantothenate) is an essential trace nutrient that functions as the obligate precursor of coenzyme A (CoA). D-Pantothenic acid sodium plays key roles in myriad biological processes, including many that regulate carbohydrate, lipid, protein, and nucleic acid metabolism[1].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro	D-Pantothenic acid sodium is a precursor to coenzyme A and is primarily involved in energy production and lipid metabolism through the TCA cycle and the β-oxidation pathway, respectively[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Pantothenic acid (PTA; 3x10, 3x100, and 3x300 mg/kg) decreases Valproic acid (VPA; 300, 400, and 500 mg/kg, s.c.)-induced neural tube defects in mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Female ICR mice weighing 29-35 g[2]
Dosage:	3x10, 3x100, and 3x300 mg/kg (10 mL/kg, volume administered)
Administration:	Injected intraperitoneally (i.p.) on day 8.5 of gestation
Result:	Significantly reduced VPA (300, 400, and 500 mg/kg, s.c.)-induced exencephaly, while none of the other external malformations such as open eyelid or skeletal malformations such as fused, absent, or bifurcated ribs and fused thoracic vertebrae and fused sternebrae were reduced.

CUSTOMER VALIDATION

- Environ Sci Pollut Res Int. 2018 Feb;25(4):3765-3774.
- Int J Mol Sci. 2023 Dec 21, 25(1), 168.
- Norwegian University of Science and Technology. Department of Clinical and Molecular Medicine. 2021 Oct.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Shuai Chen, et al. Metabolomic analysis of the toxic effect of chronic exposure of cadmium on rat urine. Environ Sci Pollut Res Int. 2018 Feb;25(4):3765-3774.

[2]. M Sato, et al. Pantothenic acid decreases valproic acid-induced neural tube defects in mice (I). Teratology. 1995 Sep;52(3):143-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com
Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA