Nicotinamide N-oxide

Cat. No.: HY-101407

CAS No.: 1986-81-8

Molecular Formula: $C_6H_6N_2O_2$

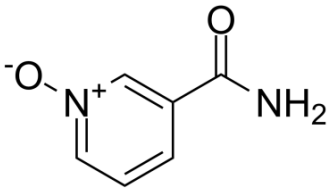
Molecular Weight: 138.12

Target: CXCR; Drug Metabolite; Endogenous Metabolite

Pathway: GPCR/G Protein; Immunology/Inflammation; Metabolic Enzyme/Protease

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : 20 mg/mL (144.80 mM; Need ultrasonic)
DMSO : 10 mg/mL (72.40 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	7.2401 mL	36.2004 mL	72.4008 mL
	5 mM	1.4480 mL	7.2401 mL	14.4802 mL
	10 mM	0.7240 mL	3.6200 mL	7.2401 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 1 mg/mL (7.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1 mg/mL (7.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Nicotinamide N-oxide, an in vivo nicotinamide metabolite, is a potent, and selective antagonist of the CXCR2 receptor.

IC_{50} & Target	CXCR2	Human Endogenous Metabolite

In Vitro

Nicotinamide is one of the forms of vitamin B_3. It is a precursor for nicotinamide adenine dinucleotide, which is best known as an electron carrier in oxidative phosphorylation and as a cofactor for many dehydrogenases. It is metabolized through two enzymatic systems. The first system starts with the methylation of nicotinamide by nicotinamide N-methyltransferase, which can subsequently be oxidized by aldehyde oxidase. The second enzymatic system oxidizes nicotinamide to nicotinamide N-oxide[1]. A series of nicotinamide N-oxides is synthesized and shown to be novel, potent, and selective

antagonists of the CXCR2 receptor. Compound 1 has demonstrated potent inhibition of neutrophil chemotaxis (IC_{50}=10 nM). Compound 2 is a selective antagonist of IL-8 binding (IC_{50}=110 nM) and potent inhibitor of neutrophil chemotaxis (IC_{50}=170 nM)[2].

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

REFERENCES

[1]. Real AM, et al. Nicotinamide N-oxidation by CYP2E1 in human liver microsomes. Drug Metab Dispos. 2013 Mar;41(3):550-3.

[2]. Cutshall NS, et al. Nicotinamide N-oxides as CXCR2 antagonists. Bioorg Med Chem Lett. 2001 Jul 23;11(14):1951-4.

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