NADPH tetrasodium salt

Cat. No.: HY-F0003

CAS No.: 2646-71-1

Molecular Formula: $C_{21}H_{26}N_7Na_4O_{17}P_3$

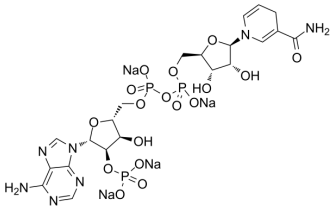
Molecular Weight: 833.35

Target: Ferroptosis; Endogenous Metabolite

Pathway: Apoptosis; Metabolic Enzyme/Protease

Storage: -20°C, stored under nitrogen

* In solvent : -80°C, 6 months; -20°C, 1 month (stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 35 mg/mL (42.00 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.2000 mL	5.9999 mL	11.9998 mL
5 mM	0.2400 mL	1.2000 mL	2.4000 mL
10 mM	0.1200 mL	0.6000 mL	1.2000 mL

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

BIOLOGICAL ACTIVITY

Description

NADPH tetrasodium salt functions as an important cofactor in a variety of metabolic and biosynthetic pathways. NADPH tetrasodium salt plays a vital role in the biosynthesis of drugs, chiral alcohols, fatty acids and biopolymers, while also being required for lipid biosynthesis, biomass formation, and cell replication. The demand for NADPH tetrasodium salt is particularly high in proliferating cancer cells, where it acts as a cofactor for the synthesis of nucleotides, proteins, and fatty acids. NADPH tetrasodium salt is also essential for the neutralization of the dangerously high levels of reactive oxygen species (ROS) generated by increased metabolic activity. NADPH tetrasodium salt is an endogenous inhibitor of ferroptosis [1][2][3][4].

IC_{50} & Target

Human Endogenous Metabolite

In Vitro

NADPH tetrasodium salt de novo synthesis is catalyzed by NAD kinases (NADKs), which catalyze the phosphorylation of NAD^+ to form $NADP^+$. NADPH tetrasodium salt is mainly involved in catabolic reactions, whereas NADPH tetrasodium salt is primarily involved in cellular antioxidative effects and anabolic reactions. NADPH tetrasodium salt is used by glutathione reductase (GR) to reduce oxidized glutathione (GSSG) to GSH[1].

The regeneration rate of NADPH tetrasodium salt is often the rate-limiting step for the over-production of desired chemicals, while maintaining robust cellular growth[2].

NADPH tetrasodium salt homeostasis is regulated by varied signaling pathways and several metabolic enzymes that undergo adaptive alteration in cancer cells. The metabolic reprogramming of NADPH tetrasodium salt renders cancer cells both highly dependent on this metabolic network for antioxidant capacity and more susceptible to oxidative stress[3]. NADPH tetrasodium salt is an essential intracellular reductant needed to eliminate lipid hydroperoxides. Indeed, NADPH tetrasodium salt levels are a biomarker of ferroptosis sensitivity across many cancer cell lines[4].

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

CUSTOMER VALIDATION

- Eur J Pharm Sci. 2021, 105889.
- Drug Des Dev Ther. 2020 Nov 30;14:5259-5273.

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REFERENCES

[1]. Tedeschi PM, et al. NAD+ Kinase as a Therapeutic Target in Cancer. Clin Cancer Res. 2016;22(21):5189-5195.

[2]. Ng CY, F et al. Rational design of a synthetic Entner-Doudoroff pathway for improved and controllable NADPH regeneration. Metab Eng. 2015;29:86-96.

[3]. Ju HQ, Lin JF, et al. NADPH homeostasis in cancer: functions, mechanisms and therapeutic implications. Signal Transduct Target Ther. 2020;5(1):231. Published 2020 Oct 7.

[4]. Stockwell BR, et al. Ferroptosis: A Regulated Cell Death Nexus Linking Metabolism, Redox Biology, and Disease. Cell. 2017;171(2):273-285.

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