

Product Data Sheet

Product Name: 6-Aminonicotinamide

Cat. No.: GC10462

Chemical Properties

Cas. No. 329-89-5

Chemical Name 6-amino-3-pyridinecarboxamide

SMILES Nc1ccc(cn1)C(=O)N

Formula $C_6H_7N_3O$

M.Wt 137.1

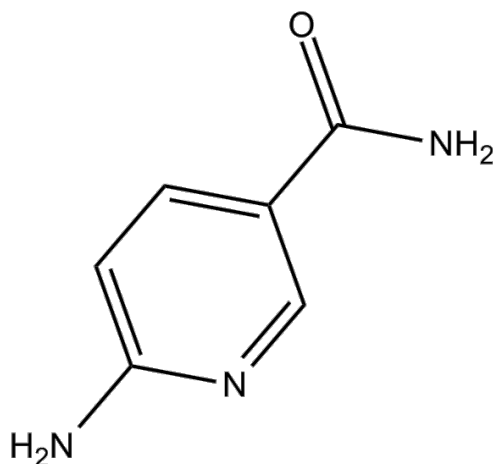
Solubility $\geq 13.7\text{mg/mL}$ in DMSO

Storage 4°C , protect from light

General tips For obtaining a higher solubility, please warm the tube at 37°C and shake it in the ultrasonic bath for a while. Stock solution can be stored below -20°C for several months.

Shipping Condition Evaluation sample solution: ship with blue ice. All other available size: ship with RT, or blue ice upon request.

Structure



Protocol

Cell experiment [1]:

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Cell lines	SJ-N-JF, NB-19, and NH-12 human neuroblastoma cells (MYCN-amplified and non-amplified)
Preparation Method	Cells were maintained in RPMI-1640 medium supplemented with 10% fetal bovine serum at 37°C, 5% CO ₂ . Cells were pretreated with 6-Aminonicotinamide at concentrations of 0.8–100µM for 18 hours, followed by 5-aminolevulinic acid (5-ALA; 250–500µM) for 6 hours and red light irradiation (633nm, 17.5mW/cm ²) for 10–15 minutes.
Reaction Conditions	0.8-100µM; 48h
Applications	6-Aminonicotinamide synergistically enhanced 5-ALA-mediated photodynamic therapy (PDT) cytotoxicity in MYCN-amplified neuroblastoma cells. The combination significantly suppressed the glutathione redox system, reducing NADPH/NADP ⁺ and GSH/GSSG ratios, and increased intracellular protoporphyrin IX (PpIX) accumulation. This led to lipid peroxidation and necrotic cell death, as evidenced by membrane rupture and ballooning, without inducing apoptosis or ferroptosis. The combination index (CI) for 6-Aminonicotinamide and 5-ALA-PDT was <1, confirming synergism.

Animal experiment [2]:

Animal models	Pregnant white mice
Preparation Method	Pregnant mice were intraperitoneally administered a single dose of 6-Aminonicotinamide at 5–20mg/kg body weight on gestational days 7–14.

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Dosage form 5–20mg/kg; i.p.; Single injection on specific gestational days.

Applications 6-Aminonicotinamide induced dose- and time-dependent teratogenic effects in fetal mice. Skeletal malformations included vertebral defects, rib anomalies, cleft palate, micrognathia, and spina bifida.

References:

- [1] Okamura SM, Chelakkot VS, Linn Z, et al. 6-Aminonicotinamide enhances the efficacy of 5-aminolevulinic acid-mediated photodynamic therapy for neuroblastoma. *BMC Cancer*. 2025 Nov 25;25(1):1815.
- [2] Curley FJ, Ingalls TH, Zappasodi P. 6-aminonicotinamide-induced skeletal malformations in mice. *Arch Environ Health*. 1968 Mar;16(3):309-15.

Background

6-Aminonicotinamide is a potent nicotinamide antimetabolite and a competitive inhibitor of the NADP⁺-dependent enzyme glucose-6-phosphate dehydrogenase (G6PD) ($K_i=0.46\mu\text{M}$)^[1-2]. 6-Aminonicotinamide interferes with glycolysis, leading to ATP depletion, and is widely used in cancer research, such as in prostate cancer and breast cancer studies^[3-4].

In vitro, treatment of HepAD38, HepG2-NTCP, and primary human hepatocytes (PHHs) with 6-Aminonicotinamide (12.5–25 μM) for 24 hours, 6-Aminonicotinamide significantly inhibits the replication and transcription of hepatitis B virus (HBV), reduces HBsAg secretion, HBV RNA levels, and core DNA levels, and slightly decreases cccDNA^[5]. Pretreatment of MYCN-amplified neuroblastoma cells (SJ-N-JF) with 6-Aminonicotinamide (0.8–100 μM) for 18 hours, followed by 5-aminolevulinic acid (5-ALA; 250–500 μM)-mediated photodynamic therapy (PDT; 633nm, 17.5mW/cm²) for 10–15 minutes, 6-Aminonicotinamide synergistically enhances the cytotoxicity of PDT, induces necrotic cell death, inhibits glucose-6-phosphate dehydrogenase (G6PD) activity, reduces the NADPH/NADP⁺ and GSH/GSSG ratios, and disrupts glutathione redox balance^[6].

In vivo, a single intraperitoneal injection of 6-Aminonicotinamide (5–20mg/kg) in pregnant mice

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(gestational days 7–14), 6-Aminonicotinamide induces developmental abnormalities in the fetuses, including skeletal malformations, cleft palate, micrognathia, and spina bifida^[7]. Intraperitoneal injection of 6-Aminonicotinamide (50mg/kg) in newborn mice (postnatal day 1), 6-Aminonicotinamide leads to rapidly progressive hydrocephalus by day 9 after injection, accompanied by aqueductal occlusion due to vacuolization of ependymal cells and periaqueductal gliosis, resulting in ventricular dilation and neurological dysfunction^[8].

References:

- [1] Ntsala LP, Lemmerer A. 6-Amino-nicotinamide. Acta Crystallogr Sect E Struct Rep Online. 2012 Aug 1;68(Pt 8):o2449.
- [2] Johnson WJ, McColl JD. 6-Aminonicotinamide--a Potent Nicotinamide Antagonist. Science. 1955 Oct 28;122(3174):834.
- [3] Cheikh IA, Hayar B, Ghanem N, et al. Therapeutic Targeting of the Pentose Phosphate Pathway in Colorectal Cancer Using 6-Aminonicotinamide and 5-Fluorouracil. Mol Carcinog. 2025 Jul;64(7):1222-1235.
- [4] Varshney R, Dwarakanath B, Jain V. Radiosensitization by 6-aminonicotinamide and 2-deoxy-D-glucose in human cancer cells. Int J Radiat Biol. 2005 May;81(5):397-408.
- [5] Ren F, Yang X, Hu ZW, et al. Niacin analogue, 6-Aminonicotinamide, a novel inhibitor of hepatitis B virus replication and HBsAg production. EBioMedicine. 2019 Nov;49:232-246.
- [6] Okamura SM, Chelakkot VS, Linn Z, et al. 6-Aminonicotinamide enhances the efficacy of 5-aminolevulinic acid-mediated photodynamic therapy for neuroblastoma. BMC Cancer. 2025 Nov 25;25(1):1815.
- [7] Curley FJ, Ingalls TH, Zappasodi P. 6-aminonicotinamide-induced skeletal malformations in mice. Arch Environ Health. 1968 Mar;16(3):309-15.
- [8] Aikawa H, Suzuki K, Ito N, et al. 6-Aminonicotinamide-induced hydrocephalus in suckling mice. J Neuropathol Exp Neurol. 1984 Sep;43(5):511-21.

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