6-Aminonicotinamide

Cat. No.: HY-W010342

CAS No.: 329-89-5

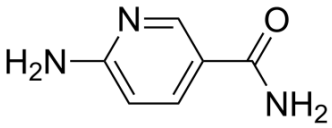
Molecular Formula: $C_6H_7N_3O$

Molecular Weight: 137.14

Target: Others

Pathway: Others

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (729.18 mM; Need ultrasonic)
H_2O : 11.11 mg/mL (81.01 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	7.2918 mL	36.4591 mL	72.9182 mL
5 mM	1.4584 mL	7.2918 mL	14.5836 mL
10 mM	0.7292 mL	3.6459 mL	7.2918 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: ≥ 2.5 mg/mL (18.23 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (18.23 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (18.23 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

6-Aminonicotinamide, a potent antimetabolite of nicotinamide, is competitive $NADP^+$-dependent enzyme glucose-6-phosphate dehydrogenase (G6PD) inhibitor (K_i=0.46 μM). 6-Aminonicotinamide interferes with glycolysis, resulting in ATP depletion and synergizes with DNA-crosslinking chemotherapy drugs, such as Cisplatin, in killing cancer cells[1][2][3][4].

In Vitro

6-Aminonicotinamide (100 nM; 7 days) causes a significant decrease in the human AR+, hormone-sensitive prostate cancer cell lines LNCaP and LAPC4, as well as the CRPC-derivative C4-2 and 22Rv1 cell models. 6-Aminonicotinamide (100 nM ± 10 nM R1881 as indicated for 3 days) increases both basal- and R1881-mediated ROS levels, suggesting 6-Aminonicotinamide is blocking the cells' antioxidant defense. 6-Aminonicotinamide also increases ROS levels in C4-2 cells[3].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	6-Aminonicotinamide (20 mg/kg; i.p.; thress times; days 1, 10 or 11, and 21) alone induces a small but significant tumor growth delay (4.3+/-0.8 days). Treatment with 6-Aminonicotinamide followed by radiation induces a tumor growth delay of 57.0+/-3.8 days in CD8F1 breast tumor model[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Cheng J, et al. TRIM21 and PHLDA3 negatively regulate the crosstalk between the PI3K/AKT pathway and PPP metabolism. Nat Commun. 2020;11(1):1880. Published 2020 Apr 20.

[2]. Almugadam SH, et al. Influence of 6-aminonicotinamide (6AN) on Leishmania promastigotes evaluated by metabolomics: Beyond the pentose phosphate pathway. Chem Biol Interact. 2018;294:167-177.

[3]. Tsouko E, et al. Regulation of the pentose phosphate pathway by an androgen receptor-mTOR-mediated mechanism and its role in prostate cancer cell growth. Oncogenesis. 2014;3(5):e103. Published 2014 May 26.

[4]. Koutcher JA, et al. Effect of 6-aminonicotinamide on the pentose phosphate pathway: 31P NMR and tumor growth delay studies. Magn Reson Med. 1996;36(6):887-892.

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