Pantoprazole

Cat. No.: HY-17507

CAS No.: 102625-70-7

Molecular Formula: $C_{16}H_{15}F_2N_3O_4S$

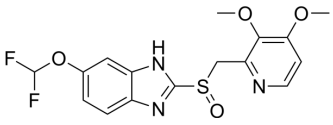
Molecular Weight: 383.37

Target: Proton Pump; Autophagy; Apoptosis; Bacterial

Pathway: Membrane Transporter/Ion Channel; Autophagy; Apoptosis; Anti-infection

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (260.84 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.6084 mL	13.0422 mL	26.0845 mL
	5 mM	0.5217 mL	2.6084 mL	5.2169 mL
	10 mM	0.2608 mL	1.3042 mL	2.6084 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 (6.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (6.52 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Pantoprazole (BY10232) is an orally active and potent proton pump inhibitor (PPI)[1]. Pantoprazole, a substituted benzimidazole, is a potent H^+/K^+-ATPase inhibitor with an IC_{50} of 6.8 μM. Pantoprazole improves pH stability and has anti-secretory, anti-ulcer activities. Pantoprazole significantly increased tumor growth delay combined with Doxorubicin (HY-15142)[3][4].

IC_{50} & Target

proton pump

In Vitro

Pantoprazole (BY1023; 1-10000 μM) leads to concentration-dependent increases in endosomal pH in EMT-6 and MCF7 cells[1]

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Pantoprazole (BY10232) can block exosome release. Pantoprazole (BY10232) inhibits the activity of V-H^+-ATPase and impaires the ability of tumour cells (melanomas, adenocarcinomas, and lymphoma cell lines) to acidify the extracellular medium[2]

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

In Vivo

Pantoprazole (BY1023; 200 mg/kg; IP; once a week for 3 weeks) significantly increases tumor growth delay of MCF-7 xenografts combined with Doxorubicin[1].

Pantoprazole (0.3-3 mg/kg, p.o.) dose-dependently decreases both basal acid secretion in pylorus-ligated rats and the stimulated acid secretion induced by mepirizole in acute fistula rats[4].

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

Animal Model:	Mice bearing MCF-7 or A431 xenografts[1]
Dosage:	200 mg/kg
Administration:	IP; once a week for 3 weeks; alone or 2 hours before Doxorubicin (6 mg/kg i.v.)
Result:	Showed even greater growth delay of MCF-7 xenografts with Doxorubicin compared with the single-dose combination. Significantly increased tumor growth delay with a single dose with Doxorubicin. There is no effect on growth delay alone.

CUSTOMER VALIDATION

- Cell Metab. 2022 Feb 7;34(3):424-440.e7.
- Nat Commun. 2023 Jul 14;14(1):4217.
- Front Oncol. 2021 Jul 7;11:660320.

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REFERENCES

[1]. Krupa J Patel, et al. Use of the proton pump inhibitor pantoprazole to modify the distribution and activity of doxorubicin: a potential strategy to improve the therapy of solid tumors. Clin Cancer Res. 2013 Dec 15;19(24):6766-76.

[2]. Huarui Zhang, et al. Advances in the discovery of exosome inhibitors in cancer. J Enzyme Inhib Med Chem. 2020 Dec;35(1):1322-1330.

[3]. W Beil, et al. Pantoprazole: a novel H+/K(+)-ATPase inhibitor with an improved pH stability. Eur J Pharmacol. 1992 Aug 6;218(2-3):265-71.

[4]. K Takeuchi, et al. Effects of pantoprazole, a novel H+/K+-ATPase inhibitor, on duodenal ulcerogenic and healing responses in rats: a comparative study with omeprazole and lansoprazole. J Gastroenterol Hepatol. 1999 Mar;14(3):251-7.

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