Pantoprazole

®

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Cat. No.:	HY-17507	
CAS No.:	102625-70-7	
Molecular Formula:	C ₁₆ H ₁₅ F ₂ N ₃ O ₄ S	-0 0-
Molecular Weight:	383.37	F, O, A, H
Target:	Proton Pump; Autophagy; Apoptosis; Bacterial	F K N
Pathway:	Membrane Transporter/Ion Channel; Autophagy; Apoptosis; Anti-infection	
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.	

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]. 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; 2 xenografts combined wi Pantoprazole (0.3-3 mg/ stimulated acid secretio MCE has not independer Animal Model:	200 mg/kg; IP; once a week for 3 weeks) significantly increases tumor growth delay of MCF-7 th Doxorubicin ^[1] . kg, p.o.) dose-dependently decreases both basal acid secretion in pylorus-ligated rats and the n induced by mepirizole in acute fistula rats ^[4] . htly confirmed the accuracy of these methods. They are for reference only. 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.

- Cell Metab. 2022 Feb 7;34(3):424-440.e7.
- 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.

Caution: Product has not been fully validated for medical applications. For research use only.

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