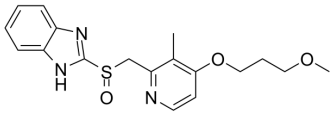
Rabeprazole

Cat. No.:	HY-B0656
CAS No.:	117976-89-3
Molecular Formula:	$C_{18}H_{21}N_3O_3S$
Molecular Weight:	359.44
Target:	Proton Pump; Apoptosis
Pathway:	Membrane Transporter/Ion Channel; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Rabeprazole (LY307640) is a second-generation proton pump inhibitor (PPI) that irreversibly inactivates gastric H^+/K^+-ATPase. Rabeprazole induces apoptosis. Rabeprazole acts as an uridine nucleoside ribohydrolase (UNH) inhibitor with an IC_{50} of 0.3 μM. Rabeprazole can be used for the research of gastric ulcerations and gastroesophageal reflux[1][2][3].

IC_{50} & Target

Pump inhibitor (PPI)[1]
IC50: 0.3 μM (UNH)[1]
H^+/K^+-ATPase[2]
Apoptosis[2]

In Vitro

Rabeprazole attenuates the cell viability of the human gastric cancer cells following treatment with 0.2 mM for 16 hours[2]. Rabeprazole completely inhibits the phosphorylation of ERK1/2 in the MKN-28 cells. The gastric cancer cell line MKN-28 is cultured in acidic culture media (pH 5.4) for 2 hours. Pretreatment with Rabeprazole (0.2 mM for 2 hours) leads to strong inhibition of ERK1/2 phosphorylation in the MKN-28 cells[2].

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

Cell Viability Assay[2]

Cell Line:	Three gastric cancer cell lines KATO III, MKN-28 and MKN-45
Concentration:	0.2 mM
Incubation Time:	16 hours
Result:	Treatment resulted in the attenuation of viability in all cancer cell lines tested, the cell viability of the MKN-28 cells significantly decreased compared with the KATO III and MKN-45 cells, respectively.

Cell Viability Assay[2]

Cell Line:	Three gastric cancer cell lines (KATO III, MKN-28 and MKN-45)[2]
Concentration:	0.2 mM
Incubation Time:	Pretreatment for 2 hours
Result:	Led to strong inhibition of ERK 1/2 phosphorylation in the MKN-28 cells, but a similar effect

	was not observed in the KATO III and MKN-45 cells.

In Vivo

Rabeprazole (10 mg/kg; P.O.; every 48 h for 18 weeks) course leads to a significant decline in bone mineral density (BMD) and decreases serum calcium level and produces secondary hyperparathyroidism in female mice[3].

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

Animal Model:	Female Swiss albino mice (body weight equals 18-26 g)[3]
Dosage:	10 mg/kg
Administration:	Oral administration; every 48 h for 18 weeks
Result:	Showed significantly lower serum calcium level compared to the vehicle treated group (5.5±2.07 vs. 9.68±2.77).

REFERENCES

[1]. Tara A Shea, et al. Identification of Proton-Pump Inhibitor Drugs That Inhibit Trichomonas Vaginalis Uridine Nucleoside Ribohydrolase. Bioorg Med Chem Lett. 2014 Feb 15;24(4):1080-4.

[2]. Aly A M Shaalan, et al. Supplement With Calcium or Alendronate Suppresses Osteopenia Due to Long Term Rabeprazole Treatment in Female Mice: Influence on Bone TRAP and Osteopontin Levels. Front Pharmacol. 2020 May 13;11:583.

[3]. Mengli Gu, et al. Rabeprazole Exhibits Antiproliferative Effects on Human Gastric Cancer Cell Lines. Oncol Lett. 2014 Oct;8(4):1739-1744.

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