Vonoprazan

Cat. No.: HY-100007

CAS No.: 881681-00-1

Molecular Formula: $C_{17}H_{16}FN_3O_2S$

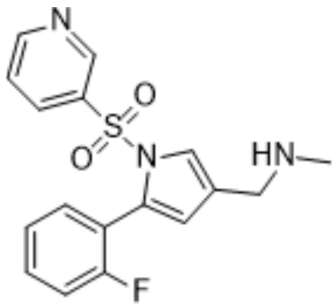
Molecular Weight: 345.39

Target: Proton Pump; Bacterial

Pathway: Membrane Transporter/Ion Channel; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (289.53 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.8953 mL	14.4764 mL	28.9528 mL
5 mM	0.5791 mL	2.8953 mL	5.7906 mL
10 mM	0.2895 mL	1.4476 mL	2.8953 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 (7.24 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.24 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vonoprazan (TAK-438 free base), a proton pump inhibitor (PPI), is a potent and orally active potassium-competitive acid blocker (P-CAB), with antisecretory activity. Vonoprazan inhibits H^+,K^+-ATPase activity in porcine gastric microsomes with an IC_{50} of 19 nM at pH 6.5. Vonoprazan is developed for the research of acid-related diseases, such as gastroesophageal reflux disease and peptic ulcer disease. Vonoprazan can be used for eradication of Helicobacter pylori[1][2][3].

IC_{50} & Target

IC50: 19 nM (porcine gastric H^+,K^+-ATPase, at pH 6.5)[2]

In Vitro	Vonoprazan (0.1 nM-10 μM; 30 minutes) exhibits porcine gastric H^+, K^+-ATPase activity in a concentration-dependent manner[2]. Vonoprazan does not inhibit Na^+,K^+-ATPase activity, even at concentrations 500 times higher than their IC_{50} values against gastric H^+,K^+-ATPase activity[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Vonoprazan (1-4 mg/kg; p.o.) completely inhibits basal and 2-deoxy-D-glucose (200 mg/kg; s.c.)-stimulated gastric acid secretion at the 4 mg/kg dose in rats[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male 7- or 8-week-old Sprague-Dawley rat[2]
Dosage:	0.5 mg/kg, 1 mg/kg, 2 mg/kg, 4 mg/kg
Administration:	Oral administration
Result:	Inhibited basal gastric acid secretion in a dose-dependent manner.

CUSTOMER VALIDATION

- Drug Metab Dispos. 2016 Oct;44(10):1543-9.
- Drug Dev Res. 2022 Dec 9.
- Br J Clin Pharmacol. 2019 Jul;85(7):1454-1463.

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REFERENCES

[1]. Sugimoto M, et al. Role of Vonoprazan in Helicobacter pylori Eradication Therapy in Japan. Front Pharmacol. 2019 Jan 15;9:1560.

[2]. Arikawa Y, et al. Discovery of a novel pyrrole derivative 1-[5-(2-fluorophenyl)-1-(pyridin-3-ylsulfonyl)-1H-pyrrol-3-yl]-N-methylmethanamine fumarate (TAK-438) as a potassium-competitive acid blocker (P-CAB). J Med Chem, 2012, 55(9), 4446-4456.

[3]. Hori Y, et al. 1-[5-(2-Fluorophenyl)-1-(pyridin-3-ylsulfonyl)-1H-pyrrol-3-yl]-N-methylmethanamine monofumarate (TAK-438), a novel and potent potassium-competitive acid blocker for the treatment of acid-related diseases. J Pharmacol Exp Ther, 2010, 335(1), 231-238.

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