Soraprazan

Cat. No.: HY-100414

CAS No.: 261944-46-1

Molecular Formula: $C_{21}H_{25}N_3O_3$

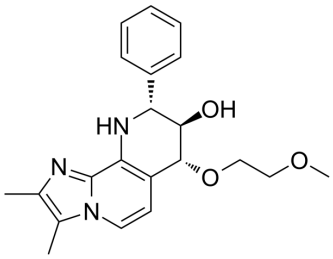
Molecular Weight: 367.44

Target: Proton Pump

Pathway: Membrane Transporter/Ion Channel

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 : 200 mg/mL (544.31 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7215 mL	13.6077 mL	27.2153 mL
	5 mM	0.5443 mL	2.7215 mL	5.4431 mL
	10 mM	0.2722 mL	1.3608 mL	2.7215 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: ≥ 5 mg/mL (13.61 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Soraprazan (BYK61359) is a selective, reversible K-competitive inhibitor of the H,K-ATPase (K_i=6.4 nM), with an IC_{50} of 0.19 μM in gastric glands. Soraprazan binds to the H,K-ATPase with a Kd of 28.27 nM. Soraprazan shows immediate inhibition of acid secretion and is more than 2000-fold selective for H,K-ATPase over Na,K- and Ca-ATPases[1].

In Vitro

Soraprazan (BYK61359) is a potent inhibitor of gastric H,K-ATPase, with an IC_{50} of 0.1 μM when measured in ion-leaky vesicles in the presence of 1 mM potassium. Soraprazan (BYK61359) also effectively inhibits dibutyryl cAMP-stimulated [^{14}C]AP accumulation in isolated gastric glands with an IC_{50} of 0.19 μM (0.09-0.40 μM geometric mean from n=6 with 95% confidence limits)[1].

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

In Vivo

Soraprazan (1-27 μmol/kg; p.o.) shows rapid and consistent inhibition of acid secretion in dog [1].

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

PROTOCOL

Kinase Assay [1]

[^{3}H]Soraprazan binding studies are carried out at 20°C. In saturation experiments to determine the K_d value, ion-leaky gastric vesicles (0.01-0.02 mg/mL) are resuspended in a buffer composed of 20 mM Tris-HCl, pH 7.0, 2 mM $MgCl_2$, and 2 mM ATP (pH 7.0 by Tris) in the presence of increasing concentrations of [^{3}H]soraprazan (0.1 nM-1 μM). Nonspecific binding is determined in the presence of a 100 fold excess of unlabeled soraprazan over the concentration range of [^{3}H]soraprazan used. The enzyme suspension (1 mL) is incubated at 20°C for 30 min and rapidly filtered through a nitrocellulose membrane filter (0.45 μM) prewet with a solution composed of 20 mM Tris-HCl, pH 7.0, 10% polyethylene glycol 3350 that is placed on top of a glass fiber filter. The membrane is ished five times with 2.5 mL of a buffer composed of 20 mM Tris-HCl, pH 7.0, and 10% polyethylene glycol 3350 to remove unbound inhibitor. The membrane is put into a 20-mL scintillation vial, dimethylacetamide (0.5 mL) is added to dissolve the membrane, and 14 mL of scintillation solvent is added and counted. Binding of [^{3}H]soraprazan is determined by subtracting the nonspecific binding of [^{3}H]soraprazan, obtained in the presence of the 100-fold excess of nonradioactive soraprazan, from the amounts of [^{3}H]soraprazan bound to the membrane in the absence of the cold inhibitor.

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

CUSTOMER VALIDATION

- J Med Chem. 2022 May 23.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Simon WA, et al. Soraprazan: setting new standards in inhibition of gastric acid secretion. J Pharmacol Exp Ther. 2007 Jun;321(3):866-74. Epub 2007 Mar 16.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA