Asapiprant

Cat. No.: HY-16763

CAS No.: 932372-01-5

Molecular Formula: $C_{24}H_{27}N_3O_7S$

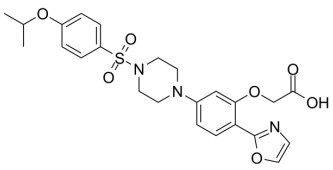
Molecular Weight: 501.55

Target: Prostaglandin Receptor

Pathway: GPCR/G Protein

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 : 11.11 mg/mL (22.15 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9938 mL	9.9691 mL	19.9382 mL
5 mM	0.3988 mL	1.9938 mL	3.9876 mL
10 mM	0.1994 mL	0.9969 mL	1.9938 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: ≥ 1.11 mg/mL (2.21 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.11 mg/mL (2.21 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.11 mg/mL (2.21 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Asapiprant is a potent and selective DP_1 receptor antagonist with a K_i of 0.44 nM.

IC_{50} & Target

DP1
0.44 nM (Ki)

In Vivo

Oral administration of Asapiprant to sheep at 1 and 3 mg/kg significantly (P<0.01) suppresses the increase in nasal resistance by 82% and 92%, respectively. Oral administration of Asapiprant to sheep at 5 mg/kg suppresses Prostaglandin

(PG) D_2 $(PGD)_2$-induced nasal resistance by 86%. When orally administered 1 h before the antigen challenge in pigs, Asapiprant (3, 10, and 30 mg/kg) suppresses immediate airway response (IAR) by 52%, 57%, and 96%, and late airway response (LAR) by 67%, 50%, and 79%, respectively. Orally administered with Asapiprant (5 mg/kg) for 4 days, the Asapiprant significantly ($P<0.01$) suppresses antigen-induced nasal resistance by 73% in sheep. Treatment with 3 and 30 mg/kg of Asapiprant in pigs significantly ($P<0.01$) suppresses nasal secretion by 53% and 72%, respectively. Treatment with Asapiprant in rats at 10 mg/kg significantly ($P<0.05$) reduces airway hyper-responsiveness (AHR), infiltration of inflammatory cells, and mucin production in bronchoalveolar lavage fluid (BALF), although treatment with Asapiprant at 0.1 mg/kg does not have a significant effect on any responses[1].

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

PROTOCOL

Animal Administration [1]

Male Hartley guinea pigs (300 to 400 g) and male brown Norway rats (240 to 400 g) are used in this study. The guinea pigs and rats are housed in an air-conditioned room at 20 to 26°C with a relative humidity of 30 to 70%, and are fed standard laboratory chow and given water ad libitum. Female sheep (32 to 46 kg) naturally sensitive to ascaris suum antigen are also used. After the oral administration of Asapiprant to rats, guinea pigs, and sheep at 10 mg/kg in suspension with 0.5% methylcellulose solution, the plasma concentrations of the drugs are measured[1].

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

REFERENCES

[1]. Takahashi G, et al. Effect of the potent and selective DP1 receptor antagonist, asapiprant (S-555739), in animal models of allergic rhinitis and allergic asthma. Eur J Pharmacol. 2015 Oct 15;765:15-23.

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