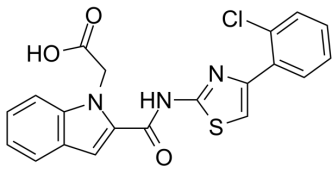
Lintitript

Cat. No.:	HY-101764
CAS No.:	136381-85-6
Molecular Formula:	$C_{20}H_{14}ClN_3O_3S$
Molecular Weight:	411.86
Target:	Cholecystokinin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (242.80 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.4280 mL	12.1400 mL	24.2801 mL
	5 mM	0.4856 mL	2.4280 mL	4.8560 mL
	10 mM	0.2428 mL	1.2140 mL	2.4280 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Lintitript (SR 27897) is a highly potent, selective, orally active, competitive and non-peptide cholecystokinin (CCK1) receptor antagonist with an EC_{50} of 6 nM and a K_i of 0.2 nM. Lintitript displays > 33-fold selectivity more selective for CCK1 than CCK2 receptors (EC_{50} value of 200 nM). Lintitript increases plasma concentration of leptin and food intake as well as plasma concentration of insulin[1][2][3].

IC_{50} & Target

EC50: 6 nM (cholecystokinin (CCK1) receptor)[2]; Ki: 0.2 nM (cholecystokinin (CCK1) receptor)[1]

In Vitro

In vitro, Lintitript (SR 27897) is a competitive antagonist of cholecystokinin (CCK)-stimulated amylase release in isolated rat pancreatic acini (pA_2 = 7.50) and of CCK-induced guinea pig gall bladder contractions (pA_2 = 9.57)[1].

Lintitript produces concentration dependent inhibition of [^{125}I]CCK binding to CCK1 receptor sites in the rat pancreas (IC_{50} value of 0.58 nM) and also to CCK 2 sites in the guinea pig cortex (IC_2 value of 479 nM). Lintitript inhibits [^{125}I]gastrin binding to gastrin receptors. Lintitript (0.5 nM) increases the dissociation constant of CCK for the CCK A receptor (K_d = 1.8 to 7.2 nM) without modifying the maximum number of receptors (B_{max} = 1800 to 1770 fmol/mg)[1].

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

In Vivo	Lintitript (SR 27897; 1 mg/kg, i.v.) completely reverses the CCK-induced amylase secretion. Lintitript also inhibits CCK-induced gastric and gallbladder emptying in mice (ED_{50}s = 3 and 72 μg/kg, respectively). Lintitript is also very active (ED_{50} = 27 μg/kg p.o.) in the gall bladder emptying protocol with egg yolk as an inducer of endogenous CCK release[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Gully D, et al. Peripheral biological activity of SR 27897: a new potent non-peptide antagonist of CCKA receptors. Eur J Pharmacol. 1993 Feb 23;232(1):13-9.

[2]. Gouldson P, et al. Contrasting roles of leu(356) in the human CCK(1) receptor for antagonist SR 27897 and agonist SR 146131 binding. Eur J Pharmacol. 1999 Nov 3;383(3):339-46.

[3]. Cano V, et al. Regulation of leptin distribution between plasma and cerebrospinal fluid by cholecystokinin receptors. Br J Pharmacol. 2003 Oct;140(4):647-52.

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