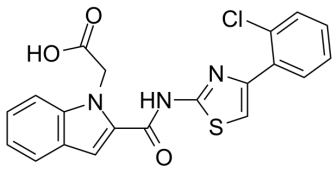
Lintitript

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



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.

Caution: Product has not been fully validated for medical applications. For research use only.

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