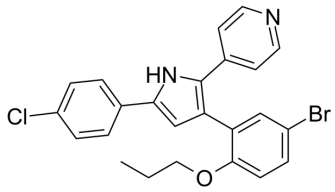
L-168049

Cat. No.:	HY-103547
CAS No.:	191034-25-0
Molecular Formula:	$C_{24}H_{20}BrClN_2O$
Molecular Weight:	467.79
Target:	GCGR
Pathway:	GPCR/G Protein
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 : ≥ 50 mg/mL (106.89 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.1377 mL	10.6886 mL	21.3771 mL
	5 mM	0.4275 mL	2.1377 mL	4.2754 mL
	10 mM	0.2138 mL	1.0689 mL	2.1377 mL

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

BIOLOGICAL ACTIVITY

Description

L-168049 is a potent, selective, orally active and non-competitive glucagon receptor antagonist with IC_{50}s of 3.7 nM, 63 nM, and 60 nM for human, murine, and canine glucagon receptors, respectively[1][2].

IC_{50} & Target

IC50: 3.7 nM (human glucagon receptor), 63 nM (murine glucagon receptor), and 60 nM (canine glucagon receptor)[2]

In Vitro

L-168049 (compound 49) inhibits glucagon (100 pM) stimulated cAMP synthesis in CHO cells expressing the human glucagon receptor (hGIAR) (IC_{50} of 41 nM). L-168049 blocks cAMP formation stimulated by glucagon in murine liver membranes[1]. L-168049 increases the apparent EC_{50} for glucagon stimulation of adenylyl cyclase in Chinese hamster ovary cells expressing the human glucagon receptor and decreases the maximal glucagon stimulation observed, with a K_b of 25 nM[2].

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

In Vivo

In the liver of L-G6pc$^{-/-}$ mice, Pck1 mRNA expression is decreased by half 6 h after the administration of L-168049 (50 mg/kg body; p.o.), demonstrating the efficiency of the suppression of glucagon signaling. In agreement with the role of glucagon in the induction of extrahepatic gluconeogenesis, the administration of the L-168049 prevents the increase of the G6pc

	expression in both the kidneys and intestine of 6 h-fasted L-G6pc$^{-/-}$ mice[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. S E de Laszlo, et al. Potent, orally absorbed glucagon receptor antagonists. Bioorg Med Chem Lett. 1999 Mar 8;9(5):641-6.

[2]. M A Cascieri, et al. Characterization of a novel, non-peptidyl antagonist of the human glucagon receptor. J Biol Chem. 1999 Mar 26;274(13):8694-7.

[3]. Elodie Mutel, et al. Control of blood glucose in the absence of hepatic glucose production during prolonged fasting in mice: induction of renal and intestinal gluconeogenesis by glucagon. Diabetes. 2011 Dec;60(12):3121-31.

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