CL097 hydrochloride

Cat. No.: HY-128799A

Molecular Formula: $C_{13}H_{15}ClN_4O$

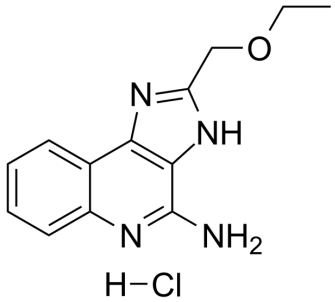
Molecular Weight: 278.74

Target: Toll-like Receptor (TLR); Reactive Oxygen Species

Pathway: Immunology/Inflammation; Metabolic Enzyme/Protease; NF-κB

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 (358.76 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.5876 mL	17.9379 mL	35.8757 mL
	5 mM	0.7175 mL	3.5876 mL	7.1751 mL
	10 mM	0.3588 mL	1.7938 mL	3.5876 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: ≥ 2.5 mg/mL (8.97 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.97 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.97 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

CL097, a potent TLR7 and TLR8 agonist, induces pro-inflammatory cytokines in macrophages[1]. CL097 induces NADPH oxidase priming, resulting in an increase of the fMLF-stimulated ROS production[2].

IC_{50} & Target	TLR7	TLR8

In Vitro

CL097 induces activation of NF-κB at 0.1 μM in TLR7 transfected HEK293 cells and at 4 μM in TLR8-transfected HEK293 cells [1].

CL097 induces hyperactivation of the NADPH oxidase by stimulating the phosphorylation of p47phox on selective sites in human neutrophils and suggest that p38 MAPK, ERK1/2, protein kinase C, and Pin1 control this process. CL097 induces the

phosphorylation of p47phox on specific sites and enhances fMLF-induced p47phox phosphorylation[2].

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

Western Blot Analysis[2]

Cell Line:	Neutrophils
Concentration:	0, 0.5, 2.5, 5, and 10 μg/mL
Incubation Time:	Pretreated for 30 minutes
Result:	Induced phosphorylation of p47phox on specific sites in a concentration-dependent manner.

In Vivo

CL097 and CD40 agonist stimulation induces efficient diabetogenic Cytotoxic T lymphocyte (CTL) function in NOD mice. CL097 (5 mg/kg, s.c.) alone causes a modest specific lysis of the target peptide (-25%). However, treatment with a combination of CL097 and CD40 agonist (10 mg/kg, i.p.) results in an increase of approximately twofold in the specific lysis of the IGRP-peptide-coated targets compared with CL097 treatment alone[3].

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

Animal Model:	Female 8.3 NOD mice (5-6 weeks old)[3]
Dosage:	5 mg/kg
Administration:	Injected s.c.
Result:	Caused a modest specific lysis of the target peptide (≈25%).

REFERENCES

[1]. Cindy Patinote, et al. Agonist and antagonist ligands of toll-like receptors 7 and 8: Ingenious tools for therapeutic purposes. Eur J Med Chem. 2020 May 1;193:112238.

[2]. Karama Makni-Maalej, et al. The TLR7/8 agonist CL097 primes N-formyl-methionyl-leucyl-phenylalanine-stimulated NADPH oxidase activation in human neutrophils: critical role of p47phox phosphorylation and the proline isomerase Pin1. J Immunol. 2012 Nov 1;18

[3]. A S Lee, et al. Toll-like receptor 7 stimulation promotes autoimmune diabetes in the NOD mouse. Diabetologia. 2011 Jun;54(6):1407-16.

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