HCH6-1

Cat. No.: HY-101283

CAS No.: 1435265-06-7

Molecular Formula: $C_{28}H_{27}N_3O_4$

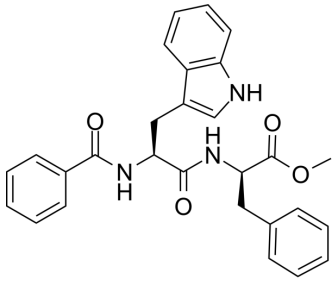
Molecular Weight: 469.53

Target: Formyl Peptide Receptor (FPR)

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 : 250 mg/mL (532.45 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1298 mL	10.6489 mL	21.2979 mL
5 mM	0.4260 mL	2.1298 mL	4.2596 mL
10 mM	0.2130 mL	1.0649 mL	2.1298 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: ≥ 2.08 mg/mL (4.43 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.43 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

HCH6-1 is a potent and competitive dipeptide antagonist of Formyl peptide receptor 1 (FPR1). HCH6-1 inhibits chemotaxis, superoxide anion generation, and elastase release in human neutrophils specifically activated by fMLF (an FPR1 agonist). HCH6-1 has protective effects against acute lung injury (ALI) in vivo and can be used for the research of FPR1-involved inflammatory lung diseases[1].

IC_{50} & Target

IC50: Formyl peptide receptor 1 (FPR1)[1]

In Vitro

In a cell-impermeable cytochrome c reduction assay, HCH6-1 significantly inhibits superoxide anion generation in fMLF (FPR1 agonist)-activated neutrophils with an IC_{50} of 0.32 μM. HCH6-1 has fewer inhibitory effects in WKYMVm (dual FPR1/FPR2 agonist)- and MMK1 (FPR2 agonist)-activated neutrophils, with IC_{50}s of 4.98±0.27 μM and 17.68±2.77 μM,

respectively[1].
HCH6-1 does not induce LDH release even at 30 μM, so it does not have cytotoxic effects in human neutrophils. HCH6-1 does not alter the level of xanthine/xanthine oxidase superoxide anion and DPPH radical in cell-free systems[1].
HCH6-1 significantly inhibits elastase release in fMLF-activated neutrophils, with an IC_{50} of 0.57 μM. However, in neutrophils triggered by WKYMVm or MMK1, HCH6-1 inhibits elastase release at higher concentrations, with IC_{50}s of 5.22±0.69 μM and 10.00±0.65 μM, respectively[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

HCH6-1 (intraperitoneal injection; 50 mg/kg; 1 h before LPS spray or 30 min after LPS spray) alone does not induce airspace inflammation. HCH6-1 pretreatment reduces inflammatory cell infiltration and distortion of pulmonary architecture in the presence of LPS. HCH6-1 posttreatment shows inhibitory effects on neutrophil accumulation and lung damage in LPS-induced ALI mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57BL/6 mice (20-25 g, 7-8 weeks old)[1]
Dosage:	50 mg/kg
Administration:	Intraperitoneal injection; 50 mg/kg; 1 h before LPS spray or 30 min after LPS spray
Result:	Ameliorated ALI in LPS-induced mice. HCH6-1-mediated decreasing of neutrophil recruitment serves as a protective mechanism in ALI mice.

CUSTOMER VALIDATION

- Cell Res. 2023 Jun 19.
- Cancer Res. 2022 Aug 16;82(16):2887-2903.

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REFERENCES

[1]. Yang SC, et al. Dipeptide HCH6-1 inhibits neutrophil activation and protects against acute lung injury by blocking FPR1. Free Radic Biol Med. 2017 May;106:254-269

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