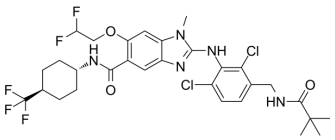
Vipoglanstat

Cat. No.:	HY-147416
CAS No.:	1360622-01-0
Molecular Formula:	$C_{30}H_{34}Cl_2F_5N_5O_3$
Molecular Weight:	678.52
Target:	PGE synthase
Pathway:	Immunology/Inflammation
Storage:	4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 150 mg/mL (221.07 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.4738 mL	7.3690 mL	14.7380 mL
	5 mM	0.2948 mL	1.4738 mL	2.9476 mL
	10 mM	0.1474 mL	0.7369 mL	1.4738 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 3.75 mg/mL (5.53 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Vipoglanstat (BI 1029539), a carboxamide, is a potent and selective, non-peptide and orally active small molecular inhibitor of human prostaglandin E synthase 1 (mPGES-1). Vipoglanstat also has anti-inflammatory activity[1][2].

In Vitro

Vipoglanstat significantly inhibits mPGES-1 level (IC_{50}: about 1 nM)[3].
Vipoglanstat blocks the up-regulation of P-gp and mPGES-1 levels on glutamate-mediatedin isolated brain capillaries[3].
Vipoglanstat reduces human peripheral blood inflammatory cell migration and inflammatory mediator release[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Vipoglanstat (30 mg/kg; i.p.) can reduce LPS-induced lung injury, with reduction in neutrophil influx, protein content, TNF-α, IL-1β and PGE2 levels in bronchoalveolar lavage (BAL), myeloperoxidase activity, expression of mPGES-1, cyclooxygenase (COX)-2 and intracellular adhesion molecule in lung tissue[2].
Vipoglanstat (30 mg/kg; p.o.; 2 h, 8 h and 22 h) significantly reduces sepsis-induced BAL inflammatory cell recruitment, lung injury score and lung expression of mPGES-1 and inducible nitric oxide synthase[2].

Vipoglanstat (30 mg/kg; p.o.; QD) also significantly prolongs survival of mice with severe sepsis[2].

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

Animal Model:	LPS-induced acute lung injury models[2]
Dosage:	30 mg/kg
Administration:	30 mg/kg, i.p.
Result:	Preserved lung architecture and reduced immune cell influx into the lungs of LPS challenged mice.

Animal Model:	CLP-induced sepsis models[2]
Dosage:	30 mg/kg
Administration:	30 mg/kg, p.o., 2 hrs, 8 hrs and 22 hrs; 30 mg/kg, p.o., QD
Result:	Attenuated CLP induced lung injury and prolongs survival.

REFERENCES

[1]. International Nonproprietary Names for Pharmaceutical Substances (INN). WHO Drug Information, Vol. 36, No. 2, 2022.

[2]. Malarvizhi Gurusamy, et al. Inhibition of microsomal prostaglandin E synthase-1 ameliorates acute lung injury in mice.

[3]. Yan-Yu Zhang, et al. Microsomal prostaglandin E 2 synthase-1 and its inhibitors: Molecular mechanisms and therapeutic significance. Pharmacol Res. 2022 Jan;175:105977.

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