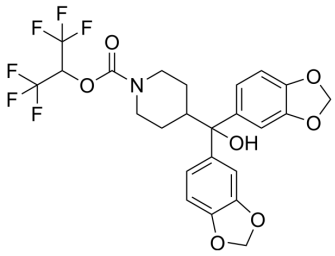
KML29

Cat. No.:	HY-18977
CAS No.:	1380424-42-9
Molecular Formula:	$C_{24}H_{21}F_6NO_7$
Molecular Weight:	549.42
Target:	MAGL
Pathway:	Metabolic Enzyme/Protease
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 : 50 mg/mL (91.01 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.8201 mL	9.1005 mL	18.2010 mL
5 mM	0.3640 mL	1.8201 mL	3.6402 mL
10 mM	0.1820 mL	0.9101 mL	1.8201 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 (4.55 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (4.55 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

KML29 is an extremely selective, orally active and irreversible MAGL inhibitor, with IC_{50} values of 15 nM, 43 nM and 5.9 nM for mouse, rat and human MAGL, respectively. KML29 exhibits minimal cross-reactivity toward other central and peripheral serine hydrolases, including no detectable activity against FAAH[1][2].

IC_{50} & Target

IC50: 15 nM (mouse MAGL), 43 nM (rat MAGL), 5.9 nM (human MAGL)[2].

In Vitro

KML29 dose-dependently elevates brain 2-AG level up to 10-fold without alteration in brain levels of anandamide, palmitoylethanolamide, and oleoylethanolamide[2].
KML29 is a potent inhibitor of 2-AG hydrolysis, but did not affect AEA hydrolysis at any concentration tested[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

KML29 enhibits antinociceptive activity without cannabimimetic side effects[3].
KML29 (20 mg/kg) has a significant but modest protective effect against LPS-induced fever[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	C57Bl/6 mice[2].
Dosage:	1-40 mg/kg.
Administration:	P.O. single dose.
Result:	Selectively inhibited MAGL in mice.

Animal Model:	Wistar albino male rats[2].
Dosage:	20 mg/kg (+LPS E. coli O111:B4 (250 μg/kg, sc)).
Administration:	SC.
Result:	Administration of KML29 simultaneously with LPS E. coli O111:B4 significantly decreased ΔT (with 5% type 1 error, 1.7 fold) compared to saline+LPS E. coli O111:B4. Administration of KML29 simultaneously with LPS E. coli O111:B4 resulted in decreased plateau phase of fever compared to LPS E. E. coli O111:B4+saline administration.

CUSTOMER VALIDATION

- Arthritis Res Ther. 2020 Jan 14;22(1):9.
- Dalhousie University. 2017 Nov.

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REFERENCES

[1]. Natsuo Ueda, et al. Discrimination between two endocannabinoids. Chem Biol. 2012 May 25;19(5):545-7.

[2]. Jae Won Chang, et al. Highly selective inhibitors of monoacylglycerol lipase bearing a reactive group that is bioisosteric with endocannabinoid substrates. Chem Biol. 2012 May 25;19(5):579-88.

[3]. B M Ignatowska-Jankowska, et al. In vivo characterization of the highly selective monoacylglycerol lipase inhibitor KML29: antinociceptive activity without cannabimimetic side effects. Br J Pharmacol. 2014 Mar;171(6):1392-407.

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

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