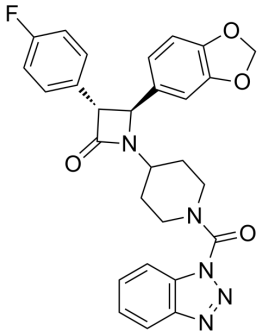
MAGL-IN-13

Cat. No.:	HY-163202
Molecular Formula:	$C_{28}H_{24}FN_5O_4$
Molecular Weight:	513.52
Target:	MAGL; Calcium Channel
Pathway:	Metabolic Enzyme/Protease; Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	MAGL-IN-13 (compound (3R, 4S) - 5v) is a selective, irreversible inhibitor for MAGL,with IC_{50} values of 0.026, 0.021 and 0.24 nM for mMAGL, hMAGL and rMAGL, respectively. MAGL-IN-13 can penetrant blood brain barrier. [1].
IC_{50} & Target	rats Ca^{2+} channel (10 μM)[1]

In Vitro

MAGL-IN-13 (preincubated for 10 min at 37℃) inhibits mMAGL, hMAGL and rMAGL with IC_{50} values of 0.026, 0.021 and 0.24 nM, respectively[1].

MAGL-IN-13 inhibits MAGL irreversibly [1].

MAGL-IN-13 (0.1 and 1 μM) reveals selectivity towards MAGL[1].

MAGL-IN-13 processes viability with IC_{50} of 47 μM and low cytoxicity potential in mouse immortalized fibroblasts NIH3T3[1].

MAGL-IN-13 inhibits rats Ca^{2+} channel at 10 μM[1].

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

Cell Cytotoxicity Assay[1]

Cell Line:	NIH 3T3
Concentration:	4, 13, 22, 34, 47, 60 μM
Incubation Time:	24 h
Result:	Exhibited low cytotoxic potential.

Cell Viability Assay[1]

Cell Line:	NIH 3T3
Concentration:	4, 13, 22, 34, 47, 60 μM
Incubation Time:	24 h
Result:	Revealed an IC_{50} of 47 μM.

In Vivo

MAGL-IN-13 (i.p.; 5 mg/kg) inhibits brain MAGL activity in a dose-dependent manner [1].

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

Animal Model:	MAGL inhibition/adult male mice [1]
Dosage:	5 mg/kg
Administration:	Intraperitoneal injection
Result:	Inhibited brain MAGL activity.

REFERENCES

[1]. Butini S et al., Development of Potent and Selective Monoacylglycerol Lipase Inhibitors. SARs, Structural Analysis, and Biological Characterization. J Med Chem. 2024 Jan 19. doi: 10.1021/acs.jmedchem.3c01278.

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