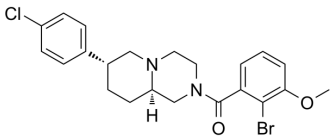
MAGLi 432

Cat. No.:	HY-150702
CAS No.:	2361575-20-2
Molecular Formula:	$C_{22}H_{24}BrClN_2O_2$
Molecular Weight:	463.8
Target:	MAGL
Pathway:	Metabolic Enzyme/Protease
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

MAGLi 432 is a non-covalent, potent, highly selective, and reversible MAGL inhibitor. MAGLi 432 binds with high affinity to the MAGL active site, with IC_{50} values of 4.2 nM (human enzyme) and 3.1 nM (mouse enzyme). MAGLi 432 can be used in the research of chronic inflammation, blood–brain barrier dysfunction, neurological disorders such as multiple sclerosis, Alzheimer's disease and Parkinson's disease[1].

In Vitro

MAGLi 432 (10 μM, 25 min) displays selectivity and potency for MAGL over other serine hydrolases in mouse and human brain lysates[1].

MAGLi 432 (1 μM, 6 h) inhibits MAGL activity and robustly enhances 2-AG levels in human NVU cells[1].

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

Western Blot Analysis[1]

Cell Line:	Human BMECs (hCMEC/D3), primary human astrocytes, and pericytes
Concentration:	10 nM, 100 nM, 1 μM and 10 μM
Incubation Time:	6 h
Result:	Inhibited MAGL activity in a dose-dependent manner, and increased 2-AG levels in all cell types. Modulated arachidonic acid levels in a cell specific-manner, with no effect in BMECs, but significant depletion in astrocytes and pericytes.

In Vivo

MAGLi 432 (intraperitoneal injection, 1 mg/kg for 3 consecutive days) inhibits MAGL in the brain and reduces arachidonic acid and PGE_2 levels in LPS-induced neuroinflammation, without reducing BBB permeability and inflammatory cytokine expression in the cortex[1].

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

Animal Model:	Male CD-1 mice model of LPS-induced neuroinflammation[1]
Dosage:	1 mg/kg for 3 consecutive days
Administration:	Intraperitoneal injection

Result:	Accumulated ~10-fold more 2-AG than vehicle controls, reducted LPS-induced PGE_2. Increased LCN2 and TNF expression compared to the LPS treatment.

REFERENCES

[1]. Alicia Kemble, et al. A potent and selective inhibitor for the modulation of MAGL activity in the neurovasculature. bioRxiv 2022.05.04.490688.

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