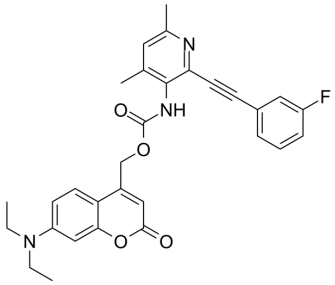
JF-NP-26

Cat. No.:	HY-131019
CAS No.:	2341841-03-8
Molecular Formula:	$C_{30}H_{28}FN_3O_4$
Molecular Weight:	513.56
Target:	mGluR
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	JF-NP-26, an inactive photocaged derivative of raseglurant, is the first caged mGlu5 receptor negative allosteric modulator. Uncaging of JF-NP-26 is elicited with light pulses in the visible spectrum (405 nm). JF-NP-26 induces light-dependent analgesia in models of inflammatory and neuropathic pain in freely behaving animals[1].
IC_{50} & Target	mGlu5 Receptor
In Vitro	The authors assessed the JF-NP-26-mediated negative allosteric modulation of mGlu5 receptor-induced responses to the orthosteric agonist quisqualate, by using an inositol phosphate (IP) accumulation assay. while JF-NP-26 didn't show activity in dark conditions, its negative allosteric modulator (NAM) activity is rescued upon 405 nm visible light illumination (pIC_{50} =7.1)[1]. Agonist challenge induced a robust mGlu5 receptor-mediated intracellular calcium rise both in dark and under 405 nm illumination, which was blocked by raseglurant. JF-NP-26 is unable to restrain agonist-mediated signalling in dark conditions, it abolished mGlu5 receptor-mediated intracellular calcium accumulation upon 405 nm irradiation, thus demonstrating a light-dependent negative allosteric modulator activity[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	JF-NP-26 (10 mg/kg; i.p.; irradiated at 405 nm (or dark) for 5 min) significantly increased pain thresholds in CCI mice only after thalamic irradiation[1]. JF-NP-26 (10 mg/kg; i.p.; at 405 nm light (or dark) for 5 min) shows light-dependent analgesic efficacy in neuropathic pain[1]. Systemic administration and in vivo photoactivation of JF-NP-26 does not impair memory in mouse[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male C57BL/6J mice weighing 20-25 g (chronic constriction injury (CCI) model)[1]
Dosage:	10 mg/kg
Administration:	I.p.; irradiated at 405 nm (or dark) for 5 min
Result:	Significantly increased pain thresholds in CCI mice only after thalamic irradiation.

Animal Model:	Formalin animal model of pain adult male CD-1 mice[1]

Dosage:	10 mg/kg
Administration:	I.p.; at 405 nm light (or dark) for 5 min
Result:	Unable to promote antinociception in dark conditions, it elicited antinociception following direct hind paw irradiation both at phase I (5 min after formalin injection in the hind paw) and phase II (20–30 min after formalin injection).

REFERENCES

[1]. Font J, et al. Optical control of pain in vivo with a photoactive mGlu5 receptor negative allosteric modulator [published correction appears in Elife. 2018 Jan 08;7:]. Elife. 2017;6:e23545. Published 2017 Apr 11.

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