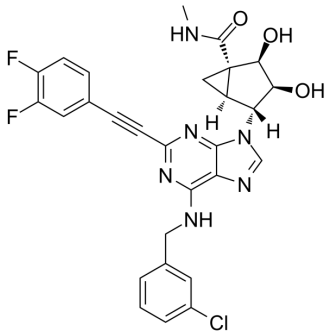
MRS5698

Cat. No.:	HY-110202
CAS No.:	1377273-00-1
Molecular Formula:	$C_{28}H_{23}ClF_2N_6O_3$
Molecular Weight:	564.97
Target:	Adenosine Receptor
Pathway:	GPCR/G Protein
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	MRS5698 is a selective G_i protein-coupled A_3 adenosine receptor (A_3AR) agonist, with K_is of approximately 3 nM for human and mouse A_3AR, respectively. MRS5698 can be used for the research of pain and psoriasis[1][2].
IC_{50} & Target	Adenosine A_3 receptor ~3 nM (Ki)
In Vitro	MRS5698 displays higher affinity and selectivity (>3000-fold) agonist A_3R vs. other adenosine receptor (ARs) in both human and mouse[1]. MRS5698 (0.1-10 μM; 1 hours) induces a concentration-dependently robust A3R-mediated cAMP reduction in HEK-293T cells permanently expressing the A3R, regardless the illumination condition[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	MRS5698 (3 nmol/day; intrathecal injection for 25 days) prevents Oxaliplatin-induced mechano-allodynia and hyperalgesia, and attenuates Oxaliplatin-induced NLRP3/IL-1β neuroinflammation[2]. MRS5698 (1 mg/kg; i.p. at days 2, 3) reduces the IL-23 induced (IL23 injected in day 0, 1, 3) ear thickness of C57BL/6N mouse during the third and fourth experimental days[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Oxaliplatin-induced Male Sprague Dawley rats (200–250 g starting weight)[2]
Dosage:	3 nmol/day
Administration:	Intrathecal injection for 25 days
Result:	Increased the value of mechanical paw withdrawal threshold in grams (PWT) in rat. Attenuated oxaliplatin-induced expression of NLRP3 and maturation of caspase 1 in the DH-SC. Reduced IL-1β levels in the spinal cord.

REFERENCES

[1]. Tosh DK, et al. Efficient, large-scale synthesis and preclinical studies of MRS5698, a highly selective A3 adenosine receptor agonist that protects against chronic

neuropathic pain. Purinergic Signal. 2015;11(3):371-387.

[2]. Wahlman C, et al. Chemotherapy-induced pain is promoted by enhanced spinal adenosine kinase levels through astrocyte-dependent mechanisms. Pain. 2018;159(6):1025-1034.

[3]. López-Cano M, et al. Optical control of adenosine A3 receptor function in psoriasis. Pharmacol Res. 2021 Aug;170:105731.

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