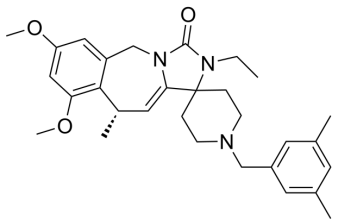
ER-819762

Cat. No.:	HY-116099
CAS No.:	1155773-15-1
Molecular Formula:	$C_{30}H_{39}N_3O_3$
Molecular Weight:	489.65
Target:	Prostaglandin Receptor
Pathway:	GPCR/G Protein
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	ER-819762 is an orally active, highly selective prostaglandin E_2 (PGE_2) EP_4 receptor antagonist with an EC_{50} of 70 nM against human EP_4 receptor. ER-819762 can be used for rheumatoid arthritis research[1].
IC_{50} & Target	EP4 70 nM (IC_{50})
In Vitro	ER-819762 suppresses human EP_4 receptor-mediated cell signalling as measured in a cAMP-dependent reporter assay (IC_{50} value of 59 ± 6 nM)[1]. ER-819762 (0-10 μM, 3 days) selectively suppresses PGE_2-induced Th1 differentiation[1]. ER-819762 (0-5 μM, 24 h) suppresses IL-23 secretion in human monocyte-derived dendritic cells[1]. ER-819762 (0.1 and 1 μM, 3 days) suppresses IL-17 production and inhibits IL-23-induced Th17 expansion in activated $CD4^+$ T cells[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	ER-819762 (0-100 mg/kg; p.o.; daily) suppresses inflammatory arthritis in mice[1]. ER-819762 (0-100 mg/kg; p.o.; once) suppresses CFA (Freund's adjuvant)-induced hyperalgesia in rat[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male DBA/1 mice, collagen-induced arthritis (CIA) model and GPI (glucose-6-phosphate isomerase)-induced arthritis model[1]
Dosage:	10, 30 and 100 mg/kg
Administration:	Oral; daily; in CIA model: from day 20 after primary immunization but before disease onset (prophylactic evaluation) or after the disease induction (therapeutic evaluation), in GPI-induced arthritis model: from day 6 after primary immunization but before disease onset (prophylactic evaluation) or after the disease induction (therapeutic evaluation).
Result:	Dose-dependently suppressed the clinical signs of arthritis and delayed disease onset, significantly suppressed disease progression when administered subsequent to the onset of disease and retarded bone erosion in the CIA model. In the GPI-induced arthritis model, significantly reduced arthritis severity and delayed disease onset when administered prior to the onset of disease and also significantly suppressed disease progression when

	administered after the establishment of arthritis.
Animal Model:	Male F344 rats, CFA-induced hyperalgesia model[1]
Dosage:	10, 30 and 100 mg/kg
Administration:	Oral, once
Result:	Significantly suppressed lame walk reaction.

REFERENCES

[1]. Chen Q, et al. A novel antagonist of the prostaglandin E(2) EP(4) receptor inhibits Th1 differentiation and Th17 expansion and is orally active in arthritis models. Br J Pharmacol. 2010 May;160(2):292-310.

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