Rolipram

Cat. No.: HY-16900

CAS No.: 61413-54-5

Molecular Formula: $C_{16}H_{21}NO_3$

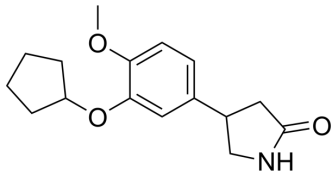
Molecular Weight: 275.34

Target: Phosphodiesterase (PDE); Bacterial; HIV

Pathway: Metabolic Enzyme/Protease; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 41 mg/mL (148.91 mM)

H_2O : < 0.1 mg/mL (ultrasonic;warming;heat to 60°C) (insoluble)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.6319 mL	18.1594 mL	36.3187 mL
5 mM	0.7264 mL	3.6319 mL	7.2637 mL
10 mM	0.3632 mL	1.8159 mL	3.6319 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (9.08 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (9.08 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.08 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Rolipram is a selective phosphodiesterases PDE4 inhibitor with IC_{50}s of 3 nM, 130 nM and 240 nM for PDE4A, PDE4B, and PDE4D, respectively.

IC_{50} & Target

PDE4

In Vitro	The PDE4 selective inhibitor, Rolipram, inhibits immunopurified PDE4B and PDE4D activities similarly, with IC_{50}s of approx. 130 nM and 240 nM respectively. In contrast, Rolipram inhibits immunopurified PDE4A activity with a dramatically lower IC_{50} of around 3 nM. Rolipram increases phosphorylation of cAMP-response-element-binding protein (CREB) in U937 cells in a dose-dependent fashion, which implies the presence of both high affinity (IC_{50} approx. 1 nM) and low affinity (IC_{50} approx. 120 nM) components. Rolipram dose-dependently inhibits the IFN-gamma-stimulated phosphorylation of p38 MAPK in a simple monotonic fashion with an IC_{50} of approx. 290 nM[1]. Rolipram is a selective PDE4 inhibitor that inhibits all PDE4 isoforms A, B, C and D. Rolipram inhibits LPS-induced TNF production in a dose-dependent manner (IC_{50} 25.9 nM), and maximal/submaximal inhibition is observed with 2 μM drug concentration in J774 cells[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	TNF mRNA and protein expression is induced by LPS in peritoneal macrophages (PM) from WT mice, and that is clearly (by 74 and 63% for TNF mRNA and TNF protein, respectively) inhibited by Rolipram. LPS-induced TNF production is enhanced in PM from MKP-1(-/-) mice as compared to that in PM from WT mice, which is in line with the published results. Interestingly, the inhibition of TNF mRNA and protein expression by Rolipram is markedly attenuated in PM from MKP-1(-/-) mice and does not reach statistical significance[2]. Repeated administration of Rolipram (1.25 mg/kg, i.p.) reduces the number of escape failures in learned helplessness rats[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	J774 murine macrophages (ATCC) are cultured at 37°C in 5% CO_2 atmosphere in DMEM supplemented with glutamax-1 containing 10% heat-inactivated FBS. For experiments, cells are seeded on 24-well plates at a density of 2×10^5 cells per well. Cell monolayers are grown for 72 h before the experiments are started. Rolipram, IBMX and BIRB 796 are dissolved in DMSO, and 8-Br-cAMP in HBSS. LPS (10 ng/mL) or the compounds of interest at concentrations indicated or the solvent (DMSO, 0.1% v/v) are added to the cells in fresh culture medium containing 10% FBS and the supplements. Cells are further incubated for the time indicated. The effect of LPS and the tested chemicals on cell viability is evaluated by Cell Proliferation Kit II (XTT)[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Inbred C57BL/6 MKP-1(-/-) mice are used. C57BL/6 mice (20-25 g) are divided into groups of six mice and treated with 200 μL of PBS or Rolipram (100 mg/kg in PBS) by an i.p. injection 2 h before applying carrageenan. Before the administration of carrageenan, the mice are anaesthetized by i.p. injection of 0.5 mg/kg of medetomidine. The mice receive a 30 μL i.d. injection of carrageenan (1.5%, dissolved in normal saline) in one hind paw. The contralateral paw receive 30 μL of saline and it is used as a control. Paw volume is measured before and 3 h after the carrageenan injection with a plethysmometer. Oedema is expressed as a change in paw volume over time. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Rep. 2021 Jul 20;36(3):109398.
- Phytomedicine. 2021, 153578.
- Antiviral Res. 2023 May 14;105635.
- Sci Signal. 2020 Nov 24;13(659):eaax0273.
- Front Pharmacol. 2018 Mar 9;9:200.

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REFERENCES

[1]. MacKenzie SJ, et al. Action of rolipram on specific PDE4 cAMP phosphodiesterase isoforms and on the phosphorylation of cAMP-response-element-binding protein (CREB) and p38 mitogen-activated protein (MAP) kinase in U937 monocyticcells. Biochem J. 2000 Apr

[2]. Korhonen R, et al. Attenuation of TNF production and experimentally induced inflammation by PDE4 inhibitor rolipram is mediated by MAPK phosphatase-1. Br J Pharmacol. 2013 Aug;169(7):1525-36.

[3]. Shalaby AM, et al. Effect of rolipram, a phosphodiesterase enzyme type-4 inhibitor, on γ-amino butyric acid content of the frontal cortex in mice exposed to chronic mild stress. J Pharmacol Pharmacother. 2012 Apr;3(2):132-7.

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