Oxaprozin

Cat. No.: HY-B0808

CAS No.: 21256-18-8

Molecular Formula: $C_{18}H_{15}NO_3$

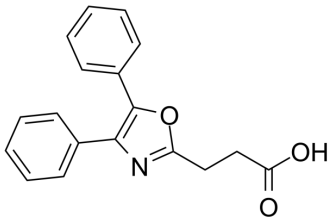
Molecular Weight: 293.32

Target: COX; NF-κB; Akt; IKK; Apoptosis

Pathway: Immunology/Inflammation; NF-κB; PI3K/Akt/mTOR; Apoptosis

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 : ≥ 100 mg/mL (340.92 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4092 mL	17.0462 mL	34.0925 mL
	5 mM	0.6818 mL	3.4092 mL	6.8185 mL
	10 mM	0.3409 mL	1.7046 mL	3.4092 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 (8.52 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (8.52 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (8.52 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Oxaprozin is an orally active and potent COX inhibitor, with IC_{50} values of 2.2 μM for human platelet COX-1 and and 36 μM for IL-1-stimulated human synovial cell COX-2, respectively. Oxaprozin also inhibits the activation of NF-κB. Oxaprozin induces cell apoptosis. Oxaprozin shows anti-inflammatory activity. Oxaprozin-mediated inhibition of the Akt/IKK/NF-κB pathway contributes to its anti-inflammatory properties[1][2].

IC_{50} & Target	COX-1	COX-2	NF-κB	IKK

	2.2 μM (IC_{50})	36 μM (IC_{50})		

In Vitro

Oxaprozin induces apoptosis in a dose-dependent manner. Oxaprozin increases caspase-3 activity in the activated but not in the resting condition. NF-κB activation is inhibited by Oxaprozin (50 μM). Oxaprozin inhibits activation of the IKK system induced by the reagent IκBα[1].

Oxaprozin (100 μM) induces the strongest proapoptotic effect and significantly increases CD40L-treated monocyte apoptosis. Oxaprozin treatment inhibits CD40L-induced Akt and NF-κB (p65) phosphorylation[2].

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

PROTOCOL

Kinase Assay [2]

Caspase 3 activity in the presence or absence of 200 ng/mL CD40L plus 1 μg/mL CD40L enhancer and 100 μM Oxaprozin is performed. The enzymatic activity is spectrophotometrically determined for 60 minutes at 405 nm assuming an extinction coefficient of 8.8×10^3 M^1/cm[2].

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

Cell Assay [2]

Purified monocytes are resuspended at 10^6/mL and cultured for 48 hours. In selective experiments, cells are cultured in the presence or absence of 50 μM PD98059, 1 μM SB203580, 50 μM LY294002, 20 μM SN-50, 50 μM Ac-DEVD-CHO, different doses (5, 10, 50, 100 μM) of Oxaprozin, 100 μM ibuprofen,100 μM indomethacin, or 100 μM naproxene. Percentages of apoptotic cells are measured by both fluorescence microscope and flow cytometer[2].

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

REFERENCES

[1]. Ottonello L, et al. Delayed apoptosis of human monocytes exposed to immune complexes is reversed byoxaprozin: role of the Akt/IkappaB kinase/nuclear factor kappaB pathway. Br J Pharmacol. 2009 May;157(2):294-306.

[2]. Montecucco F, et al. Oxaprozin-induced apoptosis on CD40 ligand-treated human primary monocytes is associated with the modulation of defined intracellular pathways. J Biomed Biotechnol. 2009;2009:478785.

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