Diclofenac

Cat. No.: HY-15036

CAS No.: 15307-86-5

Molecular Formula: $C_{14}H_{11}Cl_2NO_2$

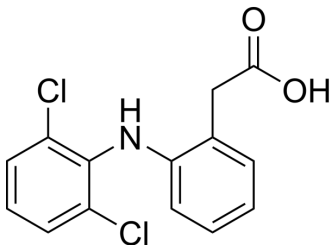
Molecular Weight: 296.15

Target: COX; Apoptosis

Pathway: Immunology/Inflammation; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	1 year
	-20°C	6 months



SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (422.08 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.3767 mL	16.8833 mL	33.7667 mL
	5 mM	0.6753 mL	3.3767 mL	6.7533 mL
	10 mM	0.3377 mL	1.6883 mL	3.3767 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: ≥ 2.08 mg/mL (7.02 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (7.02 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Diclofenac is a potent and nonselective anti-inflammatory agent, acts as a COX inhibitor, with IC_{50}s of 4 and 1.3 nM for human COX-1 and COX-2 in CHO cells[1], and 5.1 and 0.84 μM for ovine COX-1 and COX-2, respectively[2]. Diclofenac induces apoptosis of neural stem cells (NSCs) via the activation of the caspase cascade[3].

IC_{50} & Target

Human COX-2	Human COX-1	Ovine COX-2	Ovine COX-1
1.3 nM (IC_{50}, in CHO cells)	4 nM (IC_{50}, in CHO cells)	0.84 μM (IC_{50})	5.1 μM (IC_{50})

In Vitro

Diclofenac effectively blocks COX-1 mediated prostanoid production from U937 cell microsomes, with an IC_{50} of 7±3 nM[1].
Diclofenac (1-60 μM; 1 day) induces neural stem cells (NSCs)death in a concentration-dependent manner[3].
Diclofenac (10-60 μM; 6 hours) increases the expression of cleaved (activated) caspase-3[3].

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

Cell Viability Assay[3]

Cell Line:	Neural stem cells (NSCs)
Concentration:	1, 3, 10, 30, 60 μM
Incubation Time:	1 day
Result:	Induction of cell death was concentration-dependent and the effect was not saturated at a concentration of up to 60 μM.

Western Blot Analysis[3]

Cell Line:	Neural stem cells (NSCs)
Concentration:	10, 30 or 60 μM
Incubation Time:	6 hours
Result:	The activation of caspase-3 was increased in a concentration-dependent manner.

In Vivo

Diclofenac (3 mg/kg, b.i.d., for 5 days) significantly increases faecal ^{51}Cr excretion in rats, and such effect is also observed in squirrel monkeys after administrated of 1 mg/kg twice daily for 4 days[1].

Diclofenac (10 mg/kg; administered via oral route just prior to induction of inflammation) shows in vivo anti-inflammatory activity in Wistar rats[1].

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

Animal Model:	Male Sprague-Dawley rats (150±200 g)[1]
Dosage:	3 mg/kg
Administration:	Oral administration, b.i.d., for 5 days
Result:	Resulted in a significant increase in faecal ^{51}Cr excretion.

Animal Model:	Wistar rats (150-175 g) bearing Formalin-induced rat foot paw edema model[2]
Dosage:	10 mg/kg
Administration:	Administered via oral route just prior to induction of inflammation
Result:	Showed in vivo anti-inflammatory activity (% edema inhibition=29.2, 1 h; 22.2, 3 h; 20, 6 h).

CUSTOMER VALIDATION

- J Hazard Mater. 2015 May 30;289:18-27.
- Chemosphere. 2019 Jun;225:378-387.
- Int J Mol Sci. 2022, 23(20), 12066.
- Chem-Biol Interact. 2021, 109425.
- J Phys Chem Solids. 2017 October;109:117-123.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Riendeau D, et al. Biochemical and pharmacological profile of a tetrasubstituted furanone as a highly selective COX-2 inhibitor. Br J Pharmacol. 1997 May;121(1):105-17.

[2]. Labib MB, et al. Design, synthesis of novel isoindoline hybrids as COX-2 inhibitors: Anti-inflammatory, analgesic activities and docking study. Bioorg Chem. 2018 Oct;80:70-80.

[3]. Chiho Kudo, et al. Diclofenac Inhibits Proliferation and Differentiation of Neural Stem Cells. Biochem Pharmacol. 2003 Jul 15;66(2):289-95.

Caution: Product has not been fully validated for medical applications. For research use only.

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