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Lieferung & Zahlungsart

siehe unsere Liefer- und Versandbedingungen

Zuschläge

- Mindermengenzuschlag
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Diclofenac potassium

Cat. No.: HY-15038

CAS No.: 15307-81-0

Molecular Formula: $C_{14}H_{10}Cl_2KNO_2$

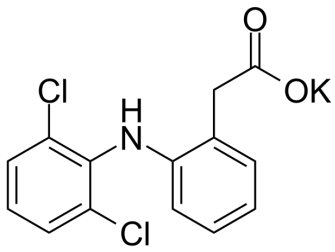
Molecular Weight: 334.24

Target: Apoptosis; COX

Pathway: Apoptosis; Immunology/Inflammation

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (299.19 mM; Need ultrasonic)

H_2O : 14.29 mg/mL (42.75 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9919 mL	14.9593 mL	29.9186 mL
5 mM	0.5984 mL	2.9919 mL	5.9837 mL
10 mM	0.2992 mL	1.4959 mL	2.9919 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 (7.48 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.48 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.48 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Diclofenac potassium 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 potassium 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 nM (IC_{50})	5.1 nM (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.

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REFERENCES

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

[2]. 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.

[3]. 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.

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