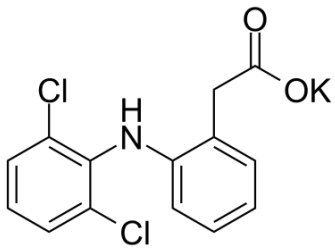
Diclofenac potassium

Cat. No.:	HY-15038
CAS No.:	15307-81-0
Molecular Formula:	$C_{14}H_{10}Cl_2KNO_2$
Molecular Weight:	334.24
Target:	COX; Apoptosis
Pathway:	Immunology/Inflammation; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



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.
- J Phys Chem Solids. 2017 October;109:117-123.
- Phys Chem Chem Phys. 2016 Jan 21;18(3):1526-36.
- Toxicol Mech Methods. 2019 Nov;29(9):654-664.

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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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