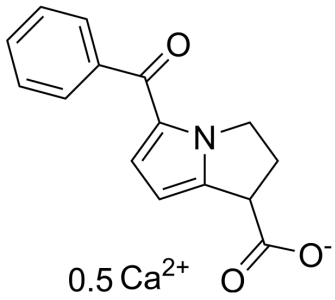
Ketorolac hemicalcium

Cat. No.:	HY-B0580C
CAS No.:	167105-81-9
Molecular Formula:	$C_{15}H_{12}CaNO_3^+$
Molecular Weight:	274.31
Target:	COX; Apoptosis
Pathway:	Immunology/Inflammation; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Ketorolac (RS37619) hemicalcium is a non-steroidal anti-inflammatory drug (NSAID), acting as a nonselective COX inhibitor, with IC_{50}s of 20 nM for COX-1 and 120 nM for COX-2. Ketorolac tromethamine is used as 0.5% ophthalmic solution for the research of allergic conjunctivitis, cystoid macular edema, intraoperative miosis, and postoperative ocular inflammation and pain. Ketorola chemicalcium is also a DDX3 inhibitor that can be used for cancer research[1][4].

In Vitro

Ketorolac (RS37619) salt (0-30 μM; 48 h) effectively kills the oral cancer cells[4].
Ketorolac salt (0-5 μM; 48 h) inhibits the expression of DDX3 protein, and induces apoptosis in H357 cells[4].
Ketorolac salt (0-2.5 μM; 0-16 h) inhibits the proliferation of oral cancer cells[4].
Ketorolac salt (0-50 μM) directly interacts with DDX3 and inhibits the ATPase activity[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[4]

Cell Line:	HOK, SCC4, SCC9 and H357 cells
Concentration:	0-30 μM
Incubation Time:	48 h
Result:	Showed inhibition with IC_{50}s of 2.6, 7.1 and 8.1 μM against H357, SCC4 and SCC9 cells, respectively. And the normal HOK cell line did not show any cell death effect.

Cell Proliferation Assay[4]

Cell Line:	H357
Concentration:	0.5, 1.0, 1.5, 2.0 and 2.5 μM
Incubation Time:	0, 8 and 16 h
Result:	Inhibited the proliferation.

Western Blot Analysis[4]

Cell Line:	H357

Concentration:	1, 2.5 and 5 μM
Incubation Time:	48 h
Result:	Significantly reduced DDX3 protein expression levels, but not completely ablated as compared to DMSO treated cells. Up regulated the expression of E-cadherin.

Apoptosis Analysis[4]

Cell Line:	H357
Concentration:	2.5 and 5 μM
Incubation Time:	48 h
Result:	Induced apoptosis.

In Vivo

Ketorolac (RS37619) (0.4% ketorolac tromethamine ophthalmic solution) shows powerful ocular anti-inflammatory activities in rabbits[1].
Ketorolac (4 mg/kg/day, p.o.; 2 weeks) has no detrimental effect in the volume fraction of bone trabeculae formed inside the alveolar socket in rats[2].
Ketorolac (60 μg; intrathecal injection; once) attenuates the damage caused by spinal cord ischemia in rats[3].
Ketorolac salt (20 and 30 mg/kg; i.p.; two times in a week for 3 weeks) reduces oral carcinogenesis in mice[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	New Zealand White rabbits (2.0–2.7 kg), LPS endotoxin-induced ocular inflammation[1]
Dosage:	50 μL ketorolac tromethamine ophthalmic solution 0.4%
Administration:	In eyes, twice, 2 hours and 1 hour before LPS challenge
Result:	Resulted in a nearly complete inhibition (98.7%) of LPS endotoxin-induced increases in FITC (fluorescein isothiocyanate)-dextran in the anterior chamber, and resulted in a nearly complete inhibition (97.5%) of LPS endotoxin-induced increases in aqueous PGE_2 concentrations in the aqueous humor.

Animal Model:	Male Wistar rats (400–450 g), spinal cord ischemia model[3]
Dosage:	30 and 60 μg
Administration:	Intrathecal injection, 1 h before the ischemia induction for once
Result:	Significantly reduced the motor disturbances and improved the survival rate at 60 μg.

Animal Model:	BALB/c mice, oral carcinogenesis model[4]
Dosage:	20 mg/kg and 30 mg/kg
Administration:	IP injection, two times in a week for 3 weeks
Result:	Decreased tumor burden, reduced expression of DDX3 and anti-apoptotic proteins (Bcl-2 and Mcl-1).

REFERENCES

[1]. Waterbury LD, et al. Comparison of cyclooxygenase inhibitory activity and ocular anti-inflammatory effects of ketorolac tromethamine and bromfenac sodium. Curr Med Res Opin. 2006 Jun;22(6):1133-40.

[2]. Fracon RN, et al. Treatment with paracetamol, ketorolac or etoricoxib did not hinder alveolar bone healing: a histometric study in rats. J Appl Oral Sci. 2010 Dec;18(6):630-4.

[3]. Hsieh YC, et al. Intrathecal ketorolac pretreatment reduced spinal cord ischemic injury in rats. Anesth Analg. 2005 Apr;100(4):1134-9.

[4]. Samal SK, et al. Ketorolac salt is a newly discovered DDX3 inhibitor to treat oral cancer. Sci Rep. 2015 Apr 28;5:9982.

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