Indomethacin sodium hydrate

Cat. No.: HY-14397A

CAS No.: 74252-25-8

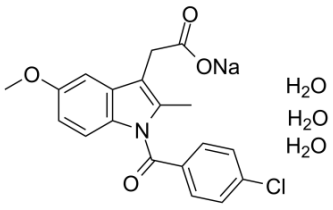
Molecular Formula: $C_{19}H_{21}ClNNaO_7$

Molecular Weight: 433.82

Target: COX; Autophagy

Pathway: Immunology/Inflammation; Autophagy

Storage: 4°C, protect from light, stored under nitrogen
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light, stored under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 33.33 mg/mL (76.83 mM; Need ultrasonic)
DMSO : 5 mg/mL (11.53 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3051 mL	11.5255 mL	23.0510 mL
	5 mM	0.4610 mL	2.3051 mL	4.6102 mL
	10 mM	0.2305 mL	1.1526 mL	2.3051 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

Indomethacin sodium hydrate (Indometacin sodium hydrate) is a potent, blood-brain permeable and nonselective inhibitor of COX1 and COX2, with IC_{50}s of 18 nM and 26 nM for human COX-1 and COX-2, respectively, in CHO cells[1]. Indomethacin sodium hydrate disrupts autophagic flux by disturbing the normal functioning of lysosomes[2].

In Vitro

Indomethacin is a potent and nonselective inhibitor of COX1 and COX2, with IC_{50}s of 18 nM and 26 nM for human COX-1 and COX-2, respectively, in CHO cells. Indomethacin inhibits lipopolysaccharide (LPS)-induced PGE2 production (COX-2) in a human whole blood assay with a potency (IC_{50}=0.68±0.17 μM), and suppresses coagulation-induced TXB2 production (COX-1) (IC_{50}=0.19±0.02 μM). Indomethacin blocks COX-1 with an IC_{50} of 20±1 nM in U937 cell microsomes at a low arachidonic acid concentration (0.1 μM)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Indomethacin dose-dependently inhibits both the carrageenan-induced rat paw oedema (ED_{50}, 2.0 mg/kg), hyperalgesia (ED_{50}, 1.5 mg/kg), and is also effective at reversing LPS-induced pyrexia in rats (ED_{50}, 1.1 mg/kg)[1]. Indomethacin (2.5 mg/kg, i.p) decreases the number of $NeuN^+$ cells in the animals at 8 days after ET-1 injection. Indomethacin also reduces microglia/macrophage activation at 14 days. Indomethacin significantly increases the number of SVZ DCX^+ cells/field at 14

days post stroke[3]. Indomethacin (22.9 mg/kg, p.o.) produces 8 to 10 linear mucosal lesions extended from the fundic to pyloric area of stomach wall[4].

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

CUSTOMER VALIDATION

- Chem Mater. 2017, 29(19):8221-8238.
- J Med Chem. 2021 Feb 23.
- Int J Nanomedicine. 2020 May 1;15:3087-3098.
- Int J Pharm. 2017 Jan 30;517(1-2):19-24.
- Langmuir. 2020 Sep 29;36(38):11374-11382.

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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]. Jorge Vallecillo-Hernández, et al. Indomethacin Disrupts Autophagic Flux by Inducing Lysosomal Dysfunction in Gastric Cancer Cells and Increases Their Sensitivity to Cytotoxic Drugs. Sci Rep. 2018 Feb 26;8(1):3593.

[3]. Lopes RS, et al. Indomethacin treatment reduces microglia activation and increases numbers of neuroblasts in the subventricular zone and ischaemic striatum after focal ischaemia. J Biosci. 2016 Sep;41(3):381-94.

[4]. Afroz S, et al. Concentrated phosphatidic acid in cereal brans as potential protective agents against indomethacin-induced stomach ulcer. J Agric Food Chem. 2016 Aug 26.

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