Ibuprofen

Cat. No.: HY-78131

CAS No.: 15687-27-1

Molecular Formula: $C_{13}H_{18}O_2$

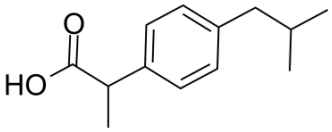
Molecular Weight: 206.28

Target: COX

Pathway: Immunology/Inflammation

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (484.78 mM)

H_2O : 1 mg/mL (4.85 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Concentration \ Solvent \ Mass	1 mg	5 mg	10 mg
1 mM	4.8478 mL	24.2389 mL	48.4778 mL
5 mM	0.9696 mL	4.8478 mL	9.6956 mL
10 mM	0.4848 mL	2.4239 mL	4.8478 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 (12.12 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (12.12 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (12.12 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ibuprofen is an anti-inflammatory agent targeting COX-1 and COX-2 with IC_{50}s of 13 μM and 370 μM, respectively.

IC_{50} & Target

COX-1	COX-2
13 μM (IC_{50})	370 μM (IC_{50})

In Vitro	Ibuprofen inhibits the enzyme cyclooxygenase COX-1 and COX-2, which convert arachidonic acid to prostaglandin H2 (PGH2). Its action is similar to aspirin, indomethacin and all other NSAIDs in intact cells, broken cells, and purified enzyme preparations[1]. Ibuprofen inhibits the constitutive activation of NF-κB and IKKα in the androgen-independent prostate tumor cells PC-3 and DU-145. It sensitizes prostate cells to ionizing radiation and blocks stimulated activation of NF-κB following exposure to TNFα or ionizing radiation in the androgen-sensitive prostate tumor cell line LNCaP. Both of these cannot be attributed directly to inhibition of IκB-α kinase but to inhibition of an upstream regulator of IKKα[2]. Ibuprofen exerts an anticancer effect by reducing survival of cancer cells. Ibuprofen is more efficacious than aspirin and acetaminophen, and comparable with (R)-flurbiprofen and indomethacin in induction of p75NTR protein expression in cell lines from bladder and other organs[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Ibuprofen reacts with the heme group of cyclooxygenase to prevent arachidonic acid conversion. Prior exposure to Ibuprofen in vivo protects cyclooxygenase completely from the irreversible effects of aspirin in platelets[4]. Ibuprofen treatment is effective in attenuating joint inflammation and early articular cartilage degeneration in the adult female Sprague-Dawley rat model induced by high-repetition and high-force (HRHF) task. It dose this by blocking the increases in serum C1 and 2C (a biomarker of collagen I and II degradation) as well as the ratio of collagen degradation to synthesis (C1, 2C/CPII, the latter a biomarker of collage type II synthesis) induced by HRHF[5]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [3]	The number of cells in each well after treatment (48 hours) with NSAIDs is estimated using the 3-(4,5-dimethylthiazol-2-yl)-2,5-diphenyltetrazolium bromide (MTT) assay. MTT labeling reagent (final concentration, 0.5 mg/mL) is added to each of the NSAID-treated T24 cells, ponasterone A alone-treated cells, ΔDDp75NTR-transfected cells plus ponasterone A, and Δ ICDp75NTR-transfected cells plus ponasterone A (2×10^3 cells/well) in 96-well culture plates (final volume, 100 µL culture medium/well) and incubated for 4 hours at 37°C in a humidified atmosphere of 10% CO_2. Subsequently, cells are incubated overnight with 100 µL of solubilization solution per well, and the samples are quantified at 570 nm using a microtiter plate reader. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [5]	At the end of the 4th week of task performance, subcohorts of the above animals are administered ibuprofen in drinking water daily (45 mg/kg body weight): NC+IBU (n=10), TR + IBU (n=11) and HRHF + IBU (n=15). HRHF+IBU animals continue to perform the HRHF task regimen with ibuprofen treatment for the remainder of the 12-week task period (i.e., an 8-week course of ibuprofen treatment). The dose used is lower than the maximum limit for gastrointestinal toxicity in rats, yet has been shown to be effective in reducing chronic inflammation. The amount of medicated water consumed/day is tracked for each animal by measuring the difference between the initial and final volume of suspended solution daily. Based on these assessments, the average weekly ibuprofen dose is similar in all groups (48.8±6.3 mg/kg body weight), with no significant differences in ibuprofen dose administered or serum levels of ibuprofen between the treated groups. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell Rep. 2019 Dec 17;29(12):3847-3858.e5.
- Chemosphere. 2019 Jun;225:378-387.
- Sci Rep. 2020 Oct 2;10(1):16383.

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REFERENCES

[1]. Noreen Y, et al. Development of a radiochemical cyclooxygenase-1 and -2 in vitro assay for identification of natural products as inhibitors of prostaglandin biosynthesis. J Nat Prod. 1998 Jan;61(1):2-7.

[2]. Palayoor ST, et al. Constitutive activation of IkappaB kinase alpha and NF-kappaB in prostate cancer cells is inhibited by ibuprofen. Oncogene. 1999 Dec 2;18(51):7389-94.

[3]. Khwaja F, et al. Ibuprofen inhibits survival of bladder cancer cells by induced expression of the p75NTR tumor suppressor protein. Cancer Res. 2004 Sep 1;64(17):6207-13.

[4]. Rao GH, et al. Ibuprofen protects platelet cyclooxygenase from irreversible inhibition by aspirin. Arteriosclerosis. 1983 Jul-Aug;3(4):383-8.

[5]. Driban JB, et al. Joint inflammation and early degeneration induced by high-force reaching are attenuated by ibuprofen in an animal model of work-related musculoskeletal disorder. J Biomed Biotechnol. 2011;2011:691412

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