

# Ibuprofen

Catalog No: tcsc1931



## Available Sizes

Size: 1g

Size: 5g



## Specifications

**CAS No:**

15687-27-1

**Formula:**

$C_{13}H_{18}O_2$

**Pathway:**

Immunology/Inflammation

**Target:**

COX

**Purity / Grade:**

>98%

**Solubility:**

H<sub>2</sub>O :

**Alternative Names:**

(±)-Ibuprofe

**Observed Molecular Weight:**

206.28

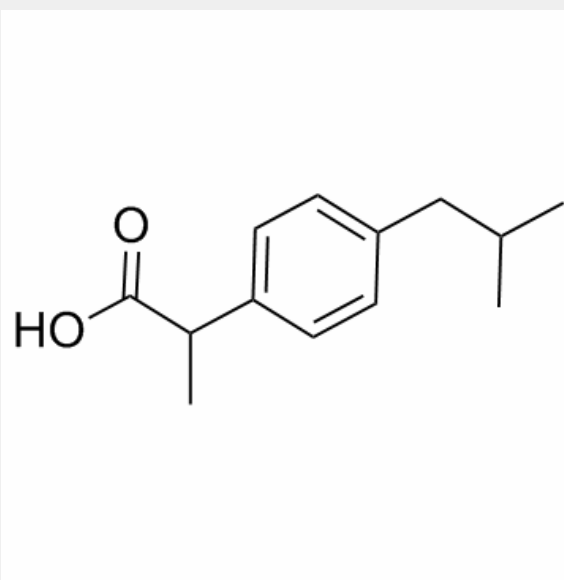
## Product Description

Ibuprofen is an anti-inflammatory inhibitor targeting **COX-1** and **COX-2** with **IC<sub>50</sub>** of 13 μM and 370 μM, respectively.

IC50 & Target: IC50: 13  $\mu$ M (COX-1), 370  $\mu$ M (COX-2)

**In Vitro:** Ibuprofen inhibits the enzyme cyclooxygenase COX-1 and COX-2, which convert arachidonic acid to prostaglandin H<sub>2</sub> (PGH<sub>2</sub>). Its action is similar to aspirin, indomethacin and all other NSAIDs in intact cells, broken cells, and purified enzyme preparations<sup>[1]</sup>. Ibuprofen inhibits the constitutive activation of NF- $\kappa$ B and IKK $\alpha$  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- $\kappa$ B following exposure to TNF $\alpha$  or ionizing radiation in the androgen-sensitive prostate tumor cell line LNCaP. Both of these cannot be attributed directly to inhibition of I $\kappa$ B- $\alpha$  kinase but to inhibition of an upstream regulator of IKK $\alpha$ <sup>[2]</sup>. 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<sup>[3]</sup>.

**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<sup>[4]</sup>. 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<sup>[5]</sup>.



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