Fenbufen

Cat. No.: HY-B1138

CAS No.: 36330-85-5

Molecular Formula: $C_{16}H_{14}O_3$

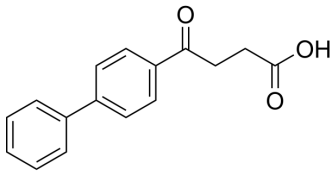
Molecular Weight: 254.28

Target: COX; Caspase

Pathway: Immunology/Inflammation; Apoptosis

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 50 mg/mL (196.63 mM; Need ultrasonic)

H_2O : < 0.1 mg/mL (insoluble)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.9327 mL	19.6634 mL	39.3267 mL
5 mM	0.7865 mL	3.9327 mL	7.8653 mL
10 mM	0.3933 mL	1.9663 mL	3.9327 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (9.83 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (9.83 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Fenbufen (CL-82204) is an orally active non-steroidal anti-inflammatory drug (NSAID), with analgetic and antipyretic effects. Fenbufen has potent activity in a variety of animal model, including carageenin edema, UV erythema and adjuvant arthritis. Fenbufen has inhibitory activities against COX-1 and COX-2 with IC_{50}s of 3.9 μM and 8.1 μM, respectively. Fenbufen is a caspases (caspase-1, 3, 4, 5, 9) inhibitor[1][2][3][4][5].

IC_{50} & Target

COX-1 3.9 μM (IC_{50})	COX-2 8.1 μM (IC_{50})	Caspase-1 4.4 μM (IC_{50})	Caspase-3 1.2 μM (IC_{50})
Caspase-4	Caspase-5	Caspase-9	

	0.57 μM (IC_{50})	0.87 μM (IC_{50})	0.76 μM (IC_{50})

In Vitro

Fenbufen (100-500 μM) improves the viability of apoptotic THP-1 cells treated with 25 μM Nigericin (HY-127019)[5].

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

In Vivo

Fenbufenmay (1200 mg/kg; feed) does not cause gastric ulceration whilst inducing a near maximal inhibition of prostaglandin release in rats[6].

Fenbufenmay (1200 mg/kg; p.o.; diet; for 10 days) blocks the hypertrophy of the heart but not that of the skeletal muscles[6].

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

Animal Model:	Male hooded Lister rats[6]
Dosage:	1200 mg/kg
Administration:	Oral administration, diet, for 10 days
Result:	Significantly reduced Clenbuterol (2mg/kg)-induced hypertrophy of the heart.

REFERENCES

[1]. A E Sloboda, et al. The pharmacological properties of fenbufen. A review. Arzneimittelforschung. 1980;30(4A):716-21.

[2]. R G Child, et al. Fenbufen, a new anti-inflammatory analgesic: synthesis and structure-activity relationships of analogs, J Pharm Sci. 1977 Apr;66(4):466-76.

[3]. A E Sloboda, et al. The pharmacology of fenbufen, 3-(4-biphenylylcarbonyl)propionic acid, and 4-biphenylacetic acid, interesting antiinflammatory-analgesic agents. Inflammation. 1976 Dec;1(4):415-38.

[4]. Asif Husain, et al. Fenbufen based 3-[5-(substituted aryl)-1,3,4-oxadiazol-2-yl]-1-(biphenyl-4-yl)propan-1-ones as safer antiinflammatory and analgesic agents. Eur J Med Chem . 2009 Sep;44(9):3798-804.

[5]. Christina E Smith, et al. Non-steroidal Anti-inflammatory Drugs Are Caspase Inhibitors. Cell Chem Biol. 2017 Mar 16;24(3):281-292.

[6]. R M Palmer, et al. Effects of the cyclo-oxygenase inhibitor, fenbufen, on clenbuterol-induced hypertrophy of cardiac and skeletal muscle of rats. Br J Pharmacol. 1990 Dec;101(4):835-8.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA