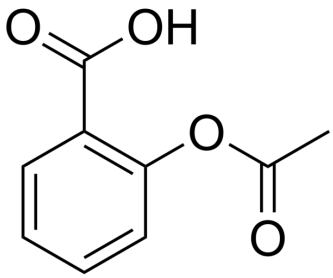
Aspirin



Cat. No.: HY-14654

CAS No.: 50-78-2

Molecular Formula: $C_9H_8O_4$

Molecular Weight: 180.16

Target: COX; Autophagy; Mitophagy; Virus Protease; Apoptosis; NF-κB; Caspase; p38 MAPK

Pathway: Immunology/Inflammation; Autophagy; Anti-infection; Apoptosis; NF-κB; MAPK/ERK Pathway

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 : 100 mg/mL (555.06 mM; Need ultrasonic)

H_2O : 3.12 mg/mL (17.32 mM; ultrasonic and warming and heat to 37°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	5.5506 mL	27.7531 mL	55.5062 mL
5 mM	1.1101 mL	5.5506 mL	11.1012 mL
10 mM	0.5551 mL	2.7753 mL	5.5506 mL

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

In Vivo

1. Add each solvent one by one: 50% PEG300 >> 50% saline
   Solubility: 5 mg/mL (27.75 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: Saline
   Solubility: 5 mg/mL (27.75 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution
5. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.88 mM); Clear solution

BIOLOGICAL ACTIVITY

Description Aspirin (Acetylsalicylic Acid) is an orally active, potent and irreversible inhibitor of cyclooxygenase COX-1 and COX-2, with IC

	$_{50}$ values of 5 and 210 μg/mL, respectively. Aspirin induces apoptosis. Aspirin inhibits the activation of NF-κB. Aspirin also inhibits platelet prostaglandin synthetase, and can prevent coronary artery and cerebrovascular thrombosis[1][2][3][4][5][6].

IC_{50} & Target	COX-1	COX-2
	27.75 μM (IC_{50})	1.17 mM (IC_{50})

In Vitro

Aspirin inhibits COX-1 and COX-2 in human articular chondrocytes, with IC_{50} values of 3.57 μM and 29.3 μM, respectively[2].
Aspirin acetylates serine-530 of COX-1, thereby blocking thromboxane A synthesis in platelets and reducing platelet aggregation[3].
Aspirin inhibits COX-2 protein expression through interference with binding of CCAAT/enhancer binding protein beta (C/EBPbeta) to its cognate site on COX-2 promoter/enhancer[3].
Aspirin inhibits NF-κB-dependent transcription from the lgκ enhancer and the human immunodeficiency virus (HIV) long terminal repeat (LTR) in transfected T cells[4].
Aspirin induces apoptosis by the activation of caspases, the activation of p38 MAP kinase, release of mitochondrial cytochrome c, and activation of the ceramide pathway[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Aspirin can be used in animal modeling to construct gastrointestinal ulcer models. Aspirin (5-150 mg/kg, PO, once) shows significant antipyretic activity in adult yeast-fevered male rats[7].

Aspirin is a classic gastric ulcer modeling agent that inhibits the synthesis of endogenous prostaglandins (PGs) in animals. Aspirin can also lyse mucosal epithelial cells phospholipids, resulting in increased mucosal permeability. Rats and mice are generally used as animal models[8][9].

Dose reference for Aspirin induction[8][9]:

(1) Model animal: Albino male mice

Gastric Ulcer Model: 500 mg/kg/day, oral, single dose

(2) Model animals: Male Wistar rats (300-350 g)

Gastric Ulcer Model: 200 mg/kg, oral, single dose

Induction of gastric Ulcer Model[8]

- Background

  Aspirin inhibits the synthesis of endogenous prostaglandins (PGs) in animals. Aspirin can also lyse mucosal epithelial cells phospholipids, resulting in increased mucosal permeability.

- Specific Mmodeling Methods

  Mice: Albino • male • 6-week-old

  Administration: 500 mg/kg • oral • single dose

- Modeling Indicators

  Behavior Observation: Caused erosionof the surface epithelial cell.

Resulted in a decrease in themucosal thickness.

Induced ulcer without COX-1reaction.

- Opposite Product(s):

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

Animal Model:	Male albino Charles River rats (200-250 g, 8 animals/group, fever was induced by 20 ml/kg of a 20 % aqueous suspension of brewer's yeast which was injected SC in the back below the nape of the neck)[7]
Dosage:	5, 25, 50, 100 and 150 mg/kg
Administration:	PO, once
Result:	Produced a statistically significant decrease of 0.23 ☒ at 15 min post-drug at the dose of 150 mg/kg. Antipyretic effect gradually increased in magnitude until a peak effect of 1.96 ☒ was reached at 120 min post-drug. The ED50 of aspirin was found to be 10.3 mg/kg with confidence limits of 1.8-23.0 mg/kg. The antipyretic response to aspirin is dependent on the dose of the compound administered.

Animal Model:	Albino male mice [8]
Dosage:	500 mg/kg, single dose
Administration:	oral
Result:	Caused erosion of the surface epithelial cells. Resulted in a decrease in the mucosal thickness. Induced ulcer without COX-1 reaction.

CUSTOMER VALIDATION

- Cell Host Microbe. 2024 Jan 11:S1931-3128(23)00510-3.
- Cancer Res. 2018 Oct 1;78(19):5586-5599.
- Cell Death Dis. 2018 Aug 28;9(9):847.
- Cell Prolif. 2022 Dec 10;e13380.
- Front Immunol. 01 December 2021.

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REFERENCES

[1]. Burch JW, et al. Inhibition of platelet prostaglandin synthetase by oral aspirin. J Clin Invest. 1978 Feb;61(2):314-9.

[2]. Elwood PC, et al. Aspirin, salicylates, and cancer. Lancet. 2009 Apr 11;373(9671):1301-9.

[3]. Loux JJ, DePalma PD, Yankell SL. Antipyretic testing of aspirin in rats. Toxicol Appl Pharmacol. 1972 Aug;22(4):672-5.

[4]. Yomna I Mahmoud, et al. Spirulina ameliorates aspirin-induced gastric ulcer in albino mice by alleviating oxidative stress and inflammation. Biomed Pharmacother. 2019.

[5]. Zhongzhi Wang, et al. Protective Effects of Ginger against Aspirin-Induced Gastric Ulcers in Rats. Yonago Acta Med. 2011, 54, 1.

[6]. Mitchell JA, et al. Selectivity of nonsteroidal antiinflammatory drugs as inhibitors of constitutive and inducible cyclooxygenase. Proc Natl Acad Sci U S A. 1993 Dec 15;90(24):11693-7.

[7]. Wu KK, et al. Aspirin and other cyclooxygenase inhibitors: new therapeutic insights. Semin Vasc Med. 2003 May;3(2):107-12.

[8]. Kopp E, et al. Inhibition of NF-kappa B by sodium salicylate and aspirin. Science. 1994 Aug 12;265(5174):956-9.

[9]. Blanco FJ, et al. Effect of antiinflammatory drugs on COX-1 and COX-2 activity in human articular chondrocytes. J Rheumatol. 1999 Jun;26(6):1366-73.

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