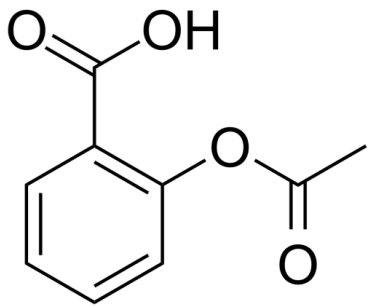
Data Sheet

Product Name:	Aspirin
Cat. No.:	CS-2001
CAS No.:	50-78-2
Molecular Formula:	C9H8O4
Molecular Weight:	180.16
Target:	Autophagy; COX; Mitophagy; Virus Protease
Pathway:	Anti-infection; Autophagy; Immunology/Inflammation
Solubility:	DMSO : 100 mg/mL (555.06 mM; Need ultrasonic); H2O : 0.1 mg/mL (0.56 mM; Need ultrasonic)



BIOLOGICAL ACTIVITY:

Aspirin is a non-selective and irreversible inhibitor of **COX-1** and **COX-2** with **IC_{50}**s of 5 and 210 μg/mL. IC50 & Target: IC50: 5 μg/mL (COX-1), 210 μg/mL (COX-2)[1] **In Vitro:** Aspirin and other non-steroid anti-inflammatory drugs inhibit the activity of cyclooxygenase (COX) which leads to the formation of prostaglandins (PGs) that cause inflammation, swelling, pain and fever[2]. Aspirin acetylates serine-530 of cyclooxygenase-1 (COX-1), thereby blocking thromboxane A synthesis in platelets and reducing platelet aggregation. This mechanism of action accounts for the effect of aspirin on prevention of coronary artery and cerebrovascular thrombosis. Aspirin is less effective in inhibiting COX-2 activity. Aspirin and salicylate inhibit 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 the activation of NF-κB. This inhibition prevents the degradation of the NF-κB inhibitor, 1κB, and therefore NF-κB is retained in the cytosol. Aspirin also 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 inhibits COX-1 and COX-2 with IC_{50} values of 3.57 μM and 29.3 μM, respectively in human articular chondrocytes[5].

PROTOCOL (Extracted from published papers and Only for reference)

Cell Assay: [5]Chondrocytes are isolated from articular cartilage of donors with no articular disease. Unstimulated and interleukin 1 (IL-1) stimulated chondrocytes are used as models to study the effects of drugs on COX-1 and COX-2. Cells are incubated with vehicle or drugs (Asprin); supernatants are removed and the level of prostaglandin E2 (PGE2) in each sample is determined by enzyme immunoassay. IC_{50}s are calculated from the reduction in PGE2 content by different concentrations of the test substance by linear regression analysis[5].

References:

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

[2]. Vane JR, et al. The mechanism of action of aspirin. Thromb Res. 2003 Jun 15;110(5-6):255-8.

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

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

[5]. 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.

CAIndexNames:

Benzoic acid, 2-(acetyloxy)-

SMILES:

OC(C1=C(OC(C)=O)C=CC=C1)=O

Caution: Product has not been fully validated for medical applications. For research use only.

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