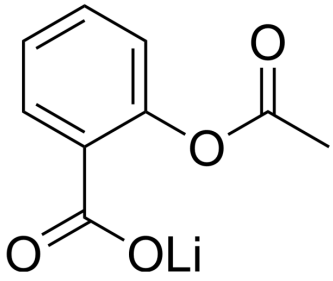
Aspirin lithium

Cat. No.:	HY-14654A
CAS No.:	552-98-7
Molecular Formula:	$C_9H_7LiO_4$
Molecular Weight:	186.09
Target:	COX; Autophagy; Virus Protease; Apoptosis; NF-κB; Mitophagy; Caspase; p38 MAPK
Pathway:	Immunology/Inflammation; Autophagy; Anti-infection; Apoptosis; NF-κB; MAPK/ERK Pathway
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Aspirin (Acetylsalicylic Acid) lithium 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 lithium induces apoptosis. Aspirin lithium inhibits the activation of NF-κB. Aspirin lithium 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

In Vitro

Aspirin lithium 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 lithium acetylates serine-530 of COX-1, thereby blocking thromboxane A synthesis in platelets and reducing platelet aggregation[3].

Aspirin lithium 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 lithium 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 lithium 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 lithium (5-150 mg/kg, PO, once) shows significant antipyretic activity in adult yeast-fevered male rats[7].

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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.

CUSTOMER VALIDATION

- Cancer Res. 2018 Oct 1;78(19):5586-5599.
- NPJ Sci Food. 2022 Dec 5;6(1):55.
- 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]. 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.

[2]. 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.

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

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

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

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