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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Salicylic acid

Cat. No.: HY-B0167

CAS No.: 69-72-7

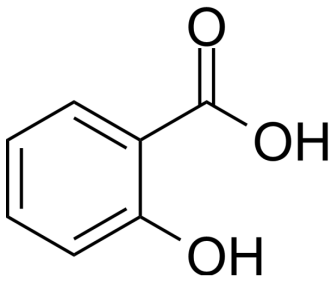
Molecular Formula: $C_7H_6O_3$

Molecular Weight: 138.12

Target: COX; Autophagy; Mitophagy; Endogenous Metabolite; Apoptosis

Pathway: Immunology/Inflammation; Autophagy; Metabolic Enzyme/Protease; Apoptosis

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 50 mg/mL (362.00 mM)

H_2O : 1 mg/mL (7.24 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Mass / Solvent / Concentration	1 mg	5 mg	10 mg
1 mM	7.2401 mL	36.2004 mL	72.4008 mL
5 mM	1.4480 mL	7.2401 mL	14.4802 mL
10 mM	0.7240 mL	3.6200 mL	7.2401 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (18.10 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (18.10 mM); Clear solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: 2.5 mg/mL (18.10 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Salicylic acid (2-Hydroxybenzoic acid) inhibits cyclo-oxygenase-2 (COX-2) activity independently of transcription factor (NF-κB) activation[1].

IC_{50} & Target

COX-2	Microbial Metabolite	Autophagy	Mitophagy
Apoptosis			

In Vitro	Salicylic acid is an effective inhibitor of COX-2 activity at concentrations far below those required to inhibit NF-κB (20 mg/mL) activation. Salicylic acid inhibits prostaglandin E_2 release when add together with interleukin 1β for 24 hr with an IC_{50} value of 5 μg/mL, an effect that is independent of NF-κB activation or COX-2 transcription or translation. Salicylic acid acutely (30 min) also causes a concentration-dependent inhibition of COX-2 activity measured in the presence of 0, 1, or 10 μM exogenous arachidonic acid. In contrast, when exogenous arachidonic acid is increased to 30 μM, Salicylic acid is a very weak inhibitor of COX-2 activity with an IC_{50} of >100 μg/mL. When added together with IL-1β for 24 hr, Salicylic acid causes a concentration-dependent inhibition of PGE_2 release with an apparent IC_{50} value of approximately 5 μg/mL. The ability of Salicylic acid to directly inhibit COX-2 activity in A549 cells is tested after a 30-min exposure period, followed by the addition of different concentrations of exogenous arachidonic acid (1, 10, and 30 μM). Salicylic acid causes a concentration-dependent inhibition of COX-2 activity in the absence of added arachidonic acid or in the presence of 1 or 10 μM exogenous substrate with an apparent IC_{50} value of approximately 5 μg/mL. However, when the same experiments are performed using 30 μM arachidonic acid, Salicylic acid is an ineffective inhibitor of COX-2 activity, with an apparent IC_{50} value of more than 100 μg/mL, and achieves a maximal inhibition of less than 50%[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	In C57Bl/6 DIO mice, Salicylic acid decreases both fasting and postprandial plasma glucose levels. Furthermore, there is a trend to reduce plasma triglyceride levels after Salicylic acid treatment in C57Bl/6 DIO mice (P=0.059). Salicylic acid significantly reduces 11β-HSD1 mRNA in omental adipose tissue in C57Bl/6 DIO mice, with a similar trend in mesenteric adipose (P=0.057). In mesenteric adipose of C57Bl/6 DIO mice, Salicylic acid also reduces 11β-HSD1 enzyme activity[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	To assess the direct effect of Salicylic acid on COX-2 activity after induction has occurred, A549 cells are first treated with IL-1β for 24 hr, and the culture medium is replaced with DMEM containing different concentrations of Salicylic acid(10, 100 and 1000 μg/mL). Cells are incubated at 37°C for 30 min. Arachidonic acid (1-30 μM) is then added for 15 min, and the medium is removed for the measurement of PGE_2[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [2]	Mice[2] Adult male C57Bl/6 mice are at age 12 weeks. Diet-induced obese C57Bl/6 mice (C57Bl/6 DIO) are given 10 weeks of high-fat diet (58% fat, 12% sucrose) before treatment. Salicylic acid (120 mg/kg/day) is administered from 1 week after arriving (C57Bl/6 Lean), after 10 weeks of high-fat feeding (C57Bl/6 DIO), or after achieving target weight (HSD1KO-DIO) for 4 weeks to groups of n=8 via osmotic minipumps implant subcutaneously between the scapulae. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Plant Cell. 2022 Aug 16;koac255.
- Cancer Lett. 2021 Jan 1;496:127-133.
- Food Chem. 2022: 134807.
- J Orthop Surg Res. 2023 Dec 15;18(1):967.
- Biopharm Drug Dispos. 2022 Oct 4.

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REFERENCES

[1]. Mitchell JA, et al. Sodium salicylate inhibits cyclo-oxygenase-2 activity independently of transcription factor (nuclear factor kappaB) activation: role of arachidonic acid. Mol Pharmacol. 1997 Jun;51(6):907-12.

[2]. Nixon M, et al. Salicylate downregulates 11β-HSD1 expression in adipose tissue in obese mice and in humans, mediating insulin sensitization. Diabetes. 2012 Apr;61(4):790-6.

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