Sodium Salicylate

Cat. No.: HY-B0167A

CAS No.: 54-21-7

Molecular Formula: $C_7H_5NaO_3$

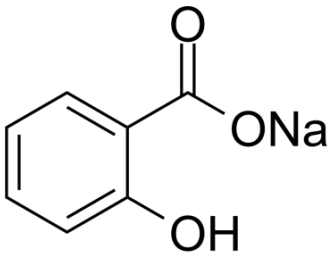
Molecular Weight: 160.1

Target: COX; NF-κB; Ribosomal S6 Kinase (RSK); Autophagy; Apoptosis

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

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 100 mg/mL (624.61 mM)

DMSO : 100 mg/mL (624.61 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	6.2461 mL	31.2305 mL	62.4610 mL
5 mM	1.2492 mL	6.2461 mL	12.4922 mL
10 mM	0.6246 mL	3.1230 mL	6.2461 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 (15.62 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (15.62 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (15.62 mM); Clear solution
4. Add each solvent one by one: PBS
   Solubility: 110 mg/mL (687.07 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Sodium Salicylate (Salicylic acid sodium salt) inhibits cyclo-oxygenase-2 (COX-2) activity independently of transcription factor (NF-κB) activation[1]. Sodium Salicylate is also a S6K inhibitor.Sodium Salicylate is a NF-κB inhibitor that decreases inflammatory gene expression and improves repair in aged muscle[4].

IC_{50} & Target	COX-2	Autophagy	S6K

In Vitro

Sodium Salicylate is an effective inhibitor of COX-2 activity at concentrations far below those required to inhibit NF-κB (20 mg/mL) activation. Sodium Salicylate 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. Sodium Salicylate 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, Sodium Salicylate 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, Sodium Salicylate causes a concentration-dependent inhibition of PGE_2 release with an apparent IC_{50} value of approximately 5 μg/mL. The ability of Sodium Salicylate 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). Sodium Salicylate 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, Sodium Salicylate 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, Salicylate decreases both fasting and postprandial plasma glucose levels. Furthermore, there is a trend to reduce plasma triglyceride levels after Salicylate treatment in C57Bl/6 DIO mice (P=0.059). Salicylate 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, Salicylate also reduces 11β-HSD1 enzyme activity[2].

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

PROTOCOL

Kinase Assay [1]

Human purified COX-2 are and the cofactors Glutathione (5 mM), Adrenaline (5 mM), and Hematin (1 μM) are dissolved in 50 mM Tris buffer (pH 7.5). Hematin is first dissolved in a concentrated stock of 100 mM in 1 M NaOH before being further diluted in Tris buffer. Enzyme reactions are carried out in individual wells of 96-well plates with a final reaction volume of 200 μL. Different concentrations of Sodium Salicylate are added to the plate, followed by the addition of 10 units of enzyme (180 μL). The plates are incubated at 37° for 30 min before Arachidonic acid (10 nM to 30 μM) is added for a further 15 min. The reaction is stopped by heating the plate to 100°C for 5 min. The 96-well plate is then centrifuged at 10,000 × g for 10 min, and appropriated samples are removed and added into the radioimmunoassay[1].

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Cell Assay [1]

To assess the direct effect of Sodium Salicylate 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 Sodium Salicylate(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].

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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. Sodium Salicylate (120 mg/kg/day) or distilled water (vehicle) 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

- Cancer Lett. 2020 Oct 8;S0304-3835(20)30506-1.
- Katedra farmakologie a toxikologie. 2020 Jul.

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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.

[3]. Dongmei Bai, et al. Palmitic acid negatively regulates tumor suppressor PTEN through T366 phosphorylation and protein degradation. Cancer Lett. 2020 Oct 8;S0304-3835(20)30506-1.

[4]. Juhyun Oh, et al. Age-associated NF-κB signaling in myofibers alters the satellite cell niche and re-strains muscle stem cell function. Aging (Albany NY). 2016 Nov; 8(11): 2871–2884.

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