Web: www.taiclone.com
Tel: +886-2-2735-9682
Email: order@taiclone.com

Salicylic acid

Catalog No: tcsc2007

Available Sizes

Size: 10g

Size: 50g

Specifications

CAS No:
69-72-7

Formula:
$C_7H_6O_3$

Pathway:
Immunology/Inflammation;Autophagy;Autophagy

Target:
COX;Autophagy;Mitophagy

Purity / Grade:
>98%

Solubility:
DMSO : ≥ 700 mg/mL (5068.06 mM)

Alternative Names:
2-Hydroxybenzoic acid

Observed Molecular Weight:
138.12

Product Description

Salicylic acid inhibits cyclo-oxygenase-2 (**COX-2**) activity independently of transcription factor (NF-κB) activation.

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IC50 & Target: COX-2[1]

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].

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].