N-Salicyloyltryptamine

Cat. No.: HY-147377

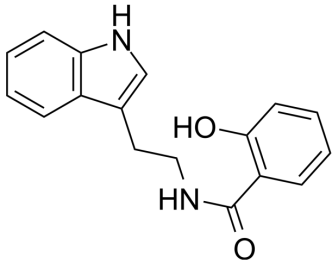
CAS No.: 31384-98-2

Molecular Formula: $C_{17}H_{16}N_2O_2$

Molecular Weight: 280.32

Target: Calcium Channel; ERK; Potassium Channel; Guanylate Cyclase; NF-κB

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling; MAPK/ERK Pathway; Stem Cell/Wnt; GPCR/G Protein; NF-κB



Storage:

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

SOLVENT & SOLUBILITY

In Vitro

DMSO : 125 mg/mL (445.92 mM; Need ultrasonic)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	3.5674 mL	17.8368 mL	35.6735 mL
5 mM	0.7135 mL	3.5674 mL	7.1347 mL
10 mM	0.3567 mL	1.7837 mL	3.5674 mL

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

BIOLOGICAL ACTIVITY

Description

N-Salicyloyltryptamine acts on voltage-dependent Na^+, Ca^{2+}, and K^+ ion channels inhibitor. N-Salicyloyltryptamine inhibits K^+ currents with an IC_{50} value of 34.6 μM (I_{to}). N-Salicyloyltryptamine also exhibits anticonvulsant, anti-inflammatory, analgesic, and vasorelaxation effect[1]-[5].

IC_{50} & Target

L-type calcium channel	potassium channel
	34.6 μM (IC_{50})

In Vitro

N-Salicyloyltryptamine (1 ng/mL-1 μg/mL; 24 h) presents no cytotoxicity and causes no oxidative stress in RAW 264.7 cells at low concentration, but (50 and 100 μg/mL) inhibits cell viability with an IC_{50} value of 22.75 μg/mL[1].

N-Salicyloyltryptamine (1 μg/mL; 24 h) reverses some redox and inflammatory parameters induced by LPS without interfering in cell viability[1].

N-Salicyloyltryptamine (1 μg/mL; 24 h) inhibits LPS-induced TNF-α and IL-1β release, as well as CD40 and TNF-α protein up-regulation[1].

N-Salicyloyltryptamine (1 μg/mL; 24 h) inhibits phosphorylation of ERK 1/2 and IκBα and p65 nuclear translocation (NF-kB

activation)[1].
N-Salicyloyltryptamine (17 μM) inhibits K^+ current by 59.27% (I_{to}) and 73.18% (I_{KD}), inhibits L-type Ca^{2+} currents by 54.9%, and shows few inhibition with high concentration (170 μM) on TTX-sensitive Na^+ current by 22.1% in GH3 cells[2].
N-Salicyloyltryptamine (0.01 nM-100 μM) produces vasorelaxation through activation of the NO/sGC/cGMP pathway and reduction of calcium influx[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	RAW 264.7 cell
Concentration:	0.001, 0.05, 1, 50, 100 μg/mL
Incubation Time:	24 hours
Result:	Resulted no effect on RAW 264.7 cell viability at 1 μg/mL; however, concentrations of 50 and 100 μg/mL significantly decreased both MTT reduction and SRB incorporation.

RT-PCR[1]

Cell Line:	RAW 264.7 cell
Concentration:	1 μg/mL
Incubation Time:	24 hours
Result:	Reduced CD40, TNF-α, and RAGE immunocontent. Inhibited ERK1/2 and IκBα phosphorylation and nuclear translocation of p65.

In Vivo

N-Salicyloyltryptamine (100 mg/kg; i.p.; 60 min before stimulation challenge) significantly inhibits pentylenetetrazol (PTZ)-induced seizures and partially eliminates the extensor reflex of maximal electric-induced seizures test[4].
N-Salicyloyltryptamine (100 mg/kg, 200 mg/kg; i.p.; single dose) shows antinociceptive and nerve excitability effects[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Swiss mice (25-35 g)[4]
Dosage:	50, 100, 200 mg/kg
Administration:	Intraperitoneal injection; single dose; 60 min before stimulation challenge
Result:	Reduced the incidence of clonic pentylenetetrazol (PTZ) seizures and mortality at 50 mg/kg, and decreased the incidence of tonic hindlimb extension (THE) produced by MES at 100, 200 mg/kg.

Animal Model:	Male Swiss mice (25-35 g)[5]
Dosage:	100 mg/kg; 200 mg/kg
Administration:	Intraperitoneal injection; single dose
Result:	Reduced the acetic acid-induced licking response of the injected paw.

REFERENCES

[1]. Gasparotto J, et al Effect of N-salicyloyltryptamine (STP), a novel tryptamine analogue, on parameters of cell viability, oxidative stress, and immunomodulation in RAW

264.7 macrophages. Cell Biol Toxicol. 2013 Jun;29(3):175-87.

[2]. Araújo DA, et al. N-salicyloyltryptamine, a new anticonvulsant drug, acts on voltage-dependent Na+, Ca2+, and K+ ion channels. Br J Pharmacol. 2003 Dec;140(7):1331-9.

[3]. Veras RC, et al. N-Salicyloyltryptamine, an N-Benzoyltryptamine Analogue, Induces Vasorelaxation through Activation of the NO/sGC Pathway and Reduction of Calcium Influx. Molecules. 2018 Jan 28;23(2):253.

[4]. Oliveira FA, et al. Anticonvulsant properties of N-salicyloyltryptamine in mice. Pharmacol Biochem Behav. 2001 Feb;68(2):199-202.

[5]. Quintans LJ Jr, et al. Bioassay-guided evaluation of antinociceptive effect of N-salicyloyltryptamine: a behavioral and electrophysiological approach. J Biomed Biotechnol. 2010;2010:230745.

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