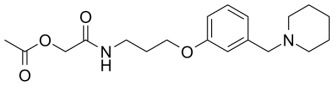
Roxatidine acetate

Cat. No.:	HY-B0305
CAS No.:	78628-28-1
Molecular Formula:	$C_{19}H_{28}N_2O_4$
Molecular Weight:	348.44
Target:	Histamine Receptor
Pathway:	GPCR/G Protein; Immunology/Inflammation; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

Roxatidine acetate is a potent, selective, competitive and orally active histamine $_{H2}$-receptor antagonist. Roxatidine acetate has antisecretory potency against gastric acid secretion. Roxatidine acetate can also suppress inflammatory responses and can be used for gastric and duodenal ulcers research. Roxatidine acetate has antitumor activity[1][2][3].

IC_{50} & Target

H_2 Receptor

In Vitro

Roxatidine acetate (0-120 μM, 1 h) suppresses inflammatory responses via inhibition of NF-κB and p38 MAPK activation in LPS-induced RAW 264.7 macrophages[2].

Roxatidine acetate (6.25 μM, 12.5 μM, and 25 μM; pre-treatment for 30 min) suppresses the PMACI-induced activation of p38 MAPK, but does not affect the phosphorylation of ERK or JNK. The total ERK 1/2, JNK, and p38 MAPK levels are unaffected by roxatidine in human mast-cells-1 (HMC-1) cells[4].

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

Western Blot Analysis[2]

Cell Line:	RAW 264.7
Concentration:	40, 80, and 120 μM
Incubation Time:	1 h
Result:	Suppressed LPS-induced PGE2, NO, and histamine production and COX-2, iNOS, and HDC expressions. Inhibited expressions of TNF-α, IL-1β, IL-6, and VEGF-1. Concentration-dependently attenuated the nuclear translocations of p65 and p50. Inhibited LPS-induced phosphorylation of p38 MAP kinase. Significantly down-regulated the LPS-induced productions of NO and PGE_2 (prostaglandin E_2).

In Vivo

Roxatidine acetate (0-300 mg/kg; p.o.; 26 days) suppressed growth of Colon 38 tumor implants in mice[3].

Roxatidine acetate (oral gavage; 20 mg/kg; single dose) inhibits Compound 48/80-increased TNF-α, IL-6, and IL-1β production and mRNA expression. Additionally, Roxatidine decreases the compound 48/80-induced degradation of procaspase-1 and appearance of the corresponding cleaved bands in mice[4].

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

Animal Model:	Male C57BL/6 Colon 38-bearing mice (8-week-old, 20 – 22 g)[3]
Dosage:	30, 100, and 300 mg/kg per day, 1 ml/100 g body weight
Administration:	Oral administration, 29 days beginning 3 days before Colon 38 implantation or 26 days beginning concomitantly with Colon 38 implantation
Result:	Suppressed growth of Colon 38 tumor implants in a dose-related manner after day 26. Suppressed VEGF levels in tumor tissue and significantly decreased serum VEGF levels.

Animal Model:	ICR male mice (6 weeks old)[4]
Dosage:	20 mg/kg
Administration:	Oral gavage; 20 mg/kg; single dose
Result:	Suppressed compound 48/80-induced allergic inflammation in anaphylactic animal model.

REFERENCES

[1]. Murdoch D, et al. Roxatidine acetate. A review of its pharmacodynamic and pharmacokinetic properties, and its therapeutic potential in peptic ulcer disease and related disorders. Drugs. 1991 Aug;42(2):240-60.

[2]. Cho EJ, et al. Roxatidine suppresses inflammatory responses via inhibition of NF-κB and p38 MAPK activation in LPS-induced RAW 264.7 macrophages. J Cell Biochem. 2011 Dec;112(12):3648-59.

[3]. Tomita K, et al. Roxatidine- and cimetidine-induced angiogenesis inhibition suppresses growth of colon cancer implants in syngeneic mice. J Pharmacol Sci. 2003 Nov;93(3):321-30.

[4]. Minho Lee, et al. Roxatidine attenuates mast cell-mediated allergic inflammation via inhibition of NF-κB and p38 MAPK activation. Sci Rep. 2017 Jan 31;7:41721.

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