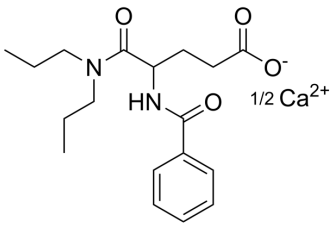
Proglumide hemicalcium

Cat. No.:	HY-103354A
CAS No.:	85068-56-0
Molecular Formula:	$C_{18}H_{26}N_2O_{4\cdot1/2}Ca$
Molecular Weight:	353.44
Target:	Cholecystokinin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Proglumide hemicalcium is a nonpeptide and orally active cholecystokinin (CCK)-A/B receptors antagonist. Proglumide hemicalcium selective blocks CCK's effects in the central nervous system (CNS). Proglumide hemicalcium has ability to inhibit gastric secretion and to protect the gastroduodenal mucosa. Proglumide hemicalcium also has antiepileptic and antioxidant activities[1][2][3][4][5].
IC_{50} & Target	Cholecystokinin (CCK)-A/B receptors[1][2]
In Vitro	In an in vitro study, Proglumide at concentrations between 0.3-10 mM inhibits CCK-stimulated amylase release dose-dependently, while Proglumide does not influence the basal amylase release at concentrations between 0-3 mM. Dose-response curves to CCK for amylase release shifted to the right with increase in Proglumide concentration. This inhibition by Proglumide is reversible. In addition, the effect of Proglumide is selective for CCK and its related peptide[2]. The incubation of HT29 cells with Proglumide significantly reduces the [^{3}H]-thymidine incorporation to HT29 cells in a dose-dependent manner, with an IC_{50} of 6.5 mM. Proglumide reduces in a dose-dependent manner the percentage of necrosis with a parallel increase of apoptosis up to 70%[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Proglumide (250-750 mg/kg; intraperitoneal injection; adult male Sprague Dawley rats) treatment is significantly effective in ameliorating the seizure activities, cognitive dysfunctions, and cerebral oxidative stress[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male Sprague Dawley rats (200-250 g; 2 months old) are induced status epilepticus (SE)[1]
Dosage:	250 mg/kg, 500 mg/kg, and 750 mg/kg
Administration:	Intraperitoneal injection
Result:	Dose-dependently and significantly increased the latencies to seizure and SE. Significantly and dose-dependently attenuated Li-PC (SE) induced increase in thiobarbituric acid (TBARS) and catalase (CAT), attenuated Li-Pc induced decrease in SOD, and attenuated depletion of GSH and glutathione-S transferase (GST) in the hippocampus and striatum.

REFERENCES

[1]. Ahmad M, et al. The effects of quinacrine, proglumide, and pentoxifylline on seizure activity, cognitive deficit, and oxidative stress in rat lithium-pilocarpine model of status epilepticus. Oxid Med Cell Longev. 2014;2014:630509.

[2]. Iwamoto Y, et al. In vitro and in vivo effect of proglumide on cholecystokinin-stimulated amylase release in mouse pancreatic acini. Gastroenterol Jpn. 1984 Feb;19(1):53-8.

[3]. González-Puga C, et al. Selective CCK-A but not CCK-B receptor antagonists inhibit HT-29 cell proliferation: synergism with pharmacological levels of melatonin. J Pineal Res. 2005 Oct;39(3):243-50.

[4]. Bunney BS, et al. Further studies on the specificity of proglumide as a selective cholecystokinin antagonist in the central nervous system. Ann N Y Acad Sci. 1985;448:345-51.

[5]. Tariq M, et al. Gastric and duodenal antiulcer and cytoprotective effects of proglumide in rats. J Pharmacol Exp Ther. 1987 May;241(2):602-7.

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