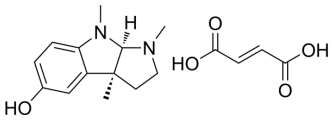
(-)-Eseroline fumarate

Cat. No.:	HY-129101
CAS No.:	70310-73-5
Molecular Formula:	$C_{17}H_{22}N_2O_5$
Molecular Weight:	334.37
Target:	5-HT Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	(-)-Eseroline fumarate is a metabolic of Physostigmine (HY-N6608), an AChE inhibitor. (-)-Eseroline fumarate elicits a leakage of lactic acid dehydrogenase (LDH) from cancer cells. (-)-Eseroline fumarate also induces the release of adenine nucleotides and 5-hydroxytryptamine (5-HT) from neuronal cells, thus induce cell death. (-)-Eseroline fumarate inhibits the electrically evoked twitches of the mouse vas deferens and of the guinea-pig ileum[1][2].
In Vitro	(-)-Eseroline fumarate (0.5 mM; 0-25 hr) time-dependently induces LDH leakage of neuronal cell culture systems: mouse neuroblastoma N1E-115, rat glioma C6, and neuroblastoma-glioma hybrid NG 108-15[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	(-)-Eseroline fumarate (HCl; 10 mg/kg; i.v.; single dose) induces the release of 5-HT from cerebral cortex in adult mongrel cats [2]. (-)-Eseroline fumarate (salicylate; 9 mg/kg; s.c.; single dose) shows analgesic effect and strongly reduces antinociceptive effect of Physostigmine (HY-N6608) in the mouse tail-flick test[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Somani SM, et al. Eseroline, a metabolite of physostigmine, induces neuronal cell death. Toxicol Appl Pharmacol. 1990 Oct;106(1):28-37.

[2]. Bartolini A, et al. Eseroline: a new antinociceptive agent derived from physostigmine with opiate receptor agonist properties. Experimental in vivo and in vitro studies on cats and rodents. Neurosci Lett. 1981 Sep 1;25(2):179-83.

[3]. Harris LS, et al. Narcotic-antagonist analgesics: interactions with cholinergic systems. J Pharmacol Exp Ther. 1969 Sep;169(1):17-22.

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