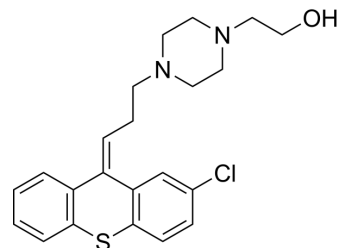


Zuclopenthixol

Cat. No.:	HY-A0163		
CAS No.:	53772-83-1		
Molecular Formula:	C ₂₂ H ₂₅ ClN ₂ OS		
Molecular Weight:	400.96		
Target:	Dopamine Receptor		
Pathway:	GPCR/G Protein; Neuronal Signaling		
Storage:	Powder	-20°C	3 years
		4°C	2 years
	In solvent	-80°C	2 years
		-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro	DMSO : 200 mg/mL (498.80 mM; Need ultrasonic)				
		Solvent Concentration	Mass 1 mg	5 mg	10 mg
	Preparing Stock Solutions	1 mM	2.4940 mL	12.4701 mL	24.9401 mL
		5 mM	0.4988 mL	2.4940 mL	4.9880 mL
10 mM		0.2494 mL	1.2470 mL	2.4940 mL	
Please refer to the solubility information to select the appropriate solvent.					
In Vivo	1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline Solubility: 2.86 mg/mL (7.13 mM); Clear solution; Need ultrasonic 2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline) Solubility: 2.86 mg/mL (7.13 mM); Suspended solution; Need ultrasonic				

BIOLOGICAL ACTIVITY

Description	Zuclopenthixol ((Z)-Clopenthixol) is a thioxanthene derivative which acts as a mixed dopamine D1/D2 receptor antagonist ^[1] [2].
IC₅₀ & Target	D ₂ Receptor
In Vivo	After acute treatment, Zuclopenthixol (0.2 and 0.4 mg/kg)-treated animals exhibit ethopharmacological profiles characterized by a decrease in offensive behaviors without impairment of motor activity (0.2 mg/kg). In contrast, the antiaggressive action of the highest dose used (0.4 mg/kg) is accompanied by a marked increase of immobility. After subchronic treatment, no tolerance to Zuclopenthixol antiaggressive or motor activity is observed ^[1] . Administration of Zuclopenthixol (0.7 and 1.4 mg/kg) significantly elevate MDA level compared to respective controls.

Nevertheless, there is no difference between the two dose levels with respect to their effect on rat brain MDA level. Post hoc pairwise comparisons between the means of groups (n=12) receiving different dose levels of Zuclopenthixol reveal that administration of 1.4 mg/kg of Zuclopenthixol significantly reduces GSH level compared to both vehicle-treated and Zuclopenthixol (0.7 mg/kg)-treated animals (P<0.001). Nevertheless, the lower dose of the drug does not affect rat brain GSH level. Animals receiving 0.7 or 1.4 mg/kg of Zuclopenthixol exhibits significantly higher GSH levels than SCO treated animals. Administration of 0.7 mg/kg of Zuclopenthixol significantly elevated GSHPx activity compared to vehicle treated animals^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration

Mice^[1]

Zuclopenthixol (0.025-0.4 mg/kg) is administered acutely or subchronically for 10 days, on agonistic behavior elicited by isolation in male mice. Individually housed mice are exposed to anosmic "standard opponents" 30 min after the drug administration, and encounters are videotaped and evaluated using an ethologically based analysis^[1].

Rats^[2]

Male albino rats of Wistar strain weighing 200-250 g are used. They are kept in a temperature of 23-25°C with alternating 12-hour light and dark cycles and allowed free access to food and water. Animals are divided into six groups (n=6). Two groups receive two dose levels of Zuclopenthixol (0.7 and 1.4 mg/kg i.p.) 60 min and SCO (1.4 mg/kg i.p.) 30 min before decapitation. A third group of rats is injected with saline, with the same content of ethanol (20% v/v) and vegetable oil (2.8% v/v) in the test solution, 60 min and then SCO (1.4 mg/kg i.p.) 30 min before decapitation. The fourth and fifth groups of rats receive two dose levels of Zuclopenthixol (0.7 and 1.4 mg/kg i.p.) 60 min and saline 30 min before decapitation. A control group of six animals is given saline, with the same content of ethanol (20% v/v) and vegetable oil (2.8% v/v) in the test solution, 60 min and then saline 30 min before decapitation and is run concurrently with drug-treated groups^[2].

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

CUSTOMER VALIDATION

- ACS Pharmacol Transl Sci. 2020 Oct 14;3(6):1278-1292.

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REFERENCES

[1]. Manzanque JM, et al. An ethopharmacological assessment of the effects of zuclopenthixol on agonistic interactions in male mice. *Methods Find Exp Clin Pharmacol*. 1999 Jan-Feb;21(1):11-5.

[2]. Khalifa AE, et al. Pro-oxidant activity of zuclopenthixol in vivo: differential effect of the drug on brain oxidative status of scopolamine-treated rats. *Hum Exp Toxicol*. 2004 Aug;23(9):439-45.

Caution: Product has not been fully validated for medical applications. For research use only.

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