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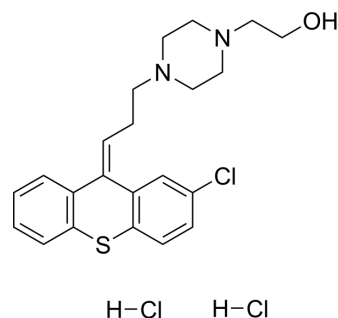
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## Zuclopenthixol dihydrochloride

<b>Cat. No.:</b>	HY-A0163B
<b>CAS No.:</b>	58045-23-1
<b>Molecular Formula:</b>	C <sub>22</sub> H <sub>27</sub> Cl <sub>3</sub> N <sub>2</sub> OS
<b>Molecular Weight:</b>	473.89
<b>Target:</b>	Dopamine Receptor
<b>Pathway:</b>	GPCR/G Protein; Neuronal Signaling
<b>Storage:</b>	4°C, sealed storage, away from moisture * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



### BIOLOGICAL ACTIVITY

<b>Description</b>	Zuclopenthixol ((Z)-Clopenthixol) dihydrochloride is a thioxanthene derivative which acts as a mixed dopamine D1/D2 receptor antagonist. Zuclopenthixol dihydrochloride is used in the study of schizophrenia <sup>[1][2][3]</sup> .
<b>IC<sub>50</sub> &amp; Target</b>	D <sub>2</sub> Receptor
<b>In Vivo</b>	<p>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<sup>[1]</sup>.</p> <p>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&lt;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<sup>[2]</sup>. MCE has not independently confirmed the accuracy of these methods. They are for reference only.</p>

### CUSTOMER VALIDATION

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

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### REFERENCES

- [1]. 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.
- [2]. Manzaneque JM, et al. An ethopharmacological assessment of the effects of zuclopenthixol on agonistic interactions in male mice. Methods Find Exp Clin Pharmacol.

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1999 Jan-Feb;21(1):11-5.

[3]. Bryan EJ, et al. Zuclopenthixol dihydrochloride for schizophrenia. Cochrane Database Syst Rev. 2017 Nov 16;11(11):CD005474.

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

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