CNQX disodium

Cat. No.: HY-15066A

CAS No.: 479347-85-8

Molecular Formula: $C_9H_2N_4Na_2O_4$

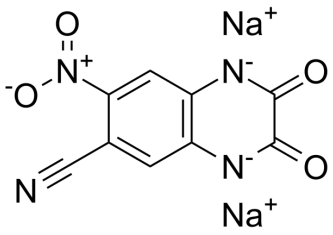
Molecular Weight: 276.12

Target: iGluR

Pathway: Membrane Transporter/Ion Channel; Neuronal Signaling

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 10.53 mg/mL (38.14 mM; ultrasonic and warming and adjust pH to 2 with HCl and heat to 70°C)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.6216 mL	18.1081 mL	36.2161 mL
	5 mM	0.7243 mL	3.6216 mL	7.2432 mL
	10 mM	0.3622 mL	1.8108 mL	3.6216 mL

Please refer to the solubility information to select the appropriate solvent.

BIOLOGICAL ACTIVITY

Description

CNQX disodium (FG9065 disodium) is a potent and competitive AMPA/kainate receptor antagonist with IC_{50}s of 0.3 μM and 1.5 μM, respectively. CNQX disodium is a competitive non-NMDA receptor antagonist[1]. CNQX disodium blocks the expression of fear-potentiated startle in rats[5].

IC_{50} & Target

IC50: 0.3 μM (AMPA) and 1.5 μM (kainate receptor)[1]

In Vitro

CNQX disodium (FG9065 disodium; 2-5 μM) reversibly blocks the Schaffer collateral and mossy fibre excitatory postsynaptic potential (EPSP), while sparing the fast and slow GABA-mediated inhibition in superfusion of hippocampal slices[2].
CNQX disodium (1-5 μM) produces a selective and dose-dependent reduction in the amplitude of the monosynaptic component of the DR-VRR recorded from lumbar spinal segments[3].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

CNQX disodium (FG9065 disodium; 0.75-3 mg/kg; IP; 20 min before testing) decreased the number of cocaine responses in a dose-dependent manner during the first 15-min cocaine-free interval[4].
The bilateral infusion of CNQX disodium (0.5 or 1.25 μg) into the amygdala or dorsal hippocampus 10 min prior to a retention test partially blocks the expression of stepdown inhibitory avoidance in rats 24 h after training. CNQX disodium causes a complete blockade at a dose of 0.5 μg[5].

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

Animal Model:	Male Wistar rats weighing 180-200 g[4]
Dosage:	0.75, 1.5, and 3 mg/kg
Administration:	IP; 20 min before testing
Result:	Decreased the number of cocaine (IV; 0.25 mg/infusion) responses in a dose-dependent manner during the first 15-min cocaine-free interval.

CUSTOMER VALIDATION

- Acta Biomater. 2022 Aug 27;S1742-7061(22)00527-X.
- Cell Death Dis. 2022 Sep 12;13(9):786.
- Biomed Pharmacother. January 2022, 112446.
- J Cell Mol Med. 2021 Aug;25(15):7342-7353.
- Front Cell Neurosci. 2019 Jun 25;13:276.

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REFERENCES

[1]. T Honoré, et al. Quinoxalinediones: Potent Competitive non-NMDA Glutamate Receptor Antagonists. Science. 1988 Aug 5;241(4866):701-3.

[2]. Alford S, et al. CNQX and DNQX block non-NMDA synaptic transmission but not NMDA-evoked locomotion in lamprey spinal cord. Brain Res. 1990 Jan 8;506(2):297-302.

[3]. Neuman RS, et al. Blockade of excitatory synaptic transmission by 6-cyano-7-nitroquinoxaline-2,3-dione(CNQX) in the hippocampus in vitro. Neurosci Lett. 1988 Sep 23;92(1):64-8.

[4]. Kim M, et al. Infusion of the non-NMDA receptor antagonist CNQX into the amygdala blocks the expression of fear-potentiated startle. Behav Neural Biol. 1993 Jan;59(1):5-8.

[5]. Pia Bäckström, et al. Attenuation of Cocaine-Seeking Behaviour by the AMPA/kainate Receptor Antagonist CNQX in Rats. Psychopharmacology (Berl). 2003 Feb;166(1):69-76.

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

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