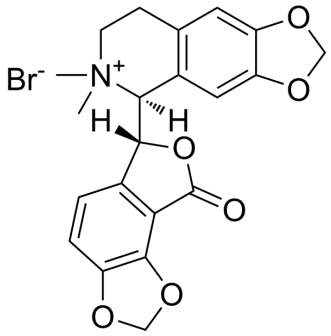
Bicuculline methobromide

Cat. No.:	HY-100783B
CAS No.:	66016-70-4
Molecular Formula:	$C_{21}H_{20}BrNO_6$
Molecular Weight:	462.29
Target:	GABA Receptor
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description	Bicuculline methobromide is a selective GABAA Receptor antagonist with an IC_{50} value of 3 μM. Bicuculline methobromide induces clonic tonic convulsions in mammals and can also be used to block Ca^{2+} activated potassium channels. Bicuculline methobromide can be used in studies of epilepsy and other related psychiatric disorders[1][2].
In Vitro	Bicuculline methobromide (1 μM and 3 μM) attains the maximal response of GABA. Bicuculline methobromide appears to shift the dose–response curves of GABA in parallel to the right without decreasing GABA maximal response, suggesting that it is a competitive antagonist at human $\alpha_1\beta_2\gamma_{2L}$ $GABA_A$ receptors expressed in Xenopus oocytes[3]. Bicuculline methobromide (1-100 μM; 2 min; applied as outside-out patches) potently blocks both Apamin (HY-P0256)-sensitive small-conductance calcium-activated potassium channels (SK2) currents and Apamin-insensitive SK1 currents in Xenopus oocytes[4]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Bicuculline methobromide (subcutaneous injection, 1.25-3 mg/kg) can cause clonus-tonic convulsions in a dose-dependent manner in mice and these convulsions are enhanced by injection of the μ-opioid agonist morphine[1]. Bicuculline methobromide (subcutaneous injection, 1.5-3.2 mg/kg) induces male Swiss S mice generalized seizures with a CD_{50} (convulsant dose) of 2.2 mg/kg for clonus and CD_{50} of 2.4 mg/kg for tonus. Seizures induced by Bicuculline at the dose of 3.2 mg/kg can be blocked by pretreatment (i.p.) with the NMDA antagonists MK-801, CPP and CGS 19755[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Cell. 2023 Mar 30;186(7):1352-1368.e18.
- Brain Behav Immun. 2023 Jun 5;S0889-1591(23)00141-1.
- Theranostics. 2022; 12(7):3057-3078.
- Cell Rep. 2021 Jul 20;36(3):109398.
- J Adv Res. 2023 Jun 21;S2090-1232(23)00171-6.

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REFERENCES

[1]. Y Yajima, et al. Effects of differential modulation of mu-, delta- and kappa-opioid systems on bicuculline-induced convulsions in the mouse. Brain Res. 2000 Apr 17;862(1-2):120-6.

[2]. W A Turski, et al. Excitatory amino acid antagonists protect mice against seizures induced by bicuculline. Brain Res. 1990 Apr 23;514(1):131-4.

[3]. Huang SH, et al. Bilobalide, a sesquiterpene trilactone from Ginkgo biloba, is an antagonist at recombinant alpha1beta2gamma2L GABA(A) receptors. Eur J Pharmacol. 2003;464(1):1-8.

[4]. Khawaled R, et al. Bicuculline block of small-conductance calcium-activated potassium channels. Pflugers Arch. 1999 Aug;438(3):314-21.

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