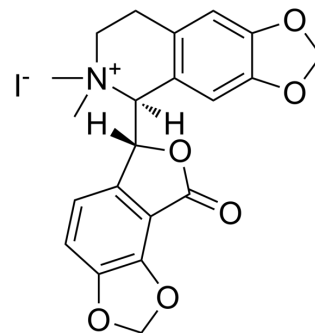


Bicuculline methiodide

| | |
|---------------------------|----------------------------------------------------------------------------------------------------------------------------------------------------|
| Cat. No.: | HY-103474 |
| CAS No.: | 40709-69-1 |
| Molecular Formula: | C ₂₁ H ₂₀ INO ₆ |
| Molecular Weight: | 509.29 |
| Target: | GABA Receptor |
| Pathway: | Membrane Transporter/Ion Channel; Neuronal Signaling |
| Storage: | 4°C, sealed storage, away from moisture and light * In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light) |



SOLVENT & SOLUBILITY

In Vitro

DMSO : 150 mg/mL (294.53 mM; Need ultrasonic)

| Concentration | Mass | | |
|---------------|-----------|-----------|------------|
| | 1 mg | 5 mg | 10 mg |
| 1 mM | 1.9635 mL | 9.8176 mL | 19.6352 mL |
| 5 mM | 0.3927 mL | 1.9635 mL | 3.9270 mL |
| 10 mM | 0.1964 mL | 0.9818 mL | 1.9635 mL |

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

BIOLOGICAL ACTIVITY

Description

Bicuculline methiodide is a potent GABA(A) receptors blocker. Bicuculline methiodide alters membrane properties and firing pattern. Bicuculline methiodide reduces the Apamin-sensitive afterhyperpolarization, while Apamin is a toxin isolated from bee venom to block small conductance Ca²⁺-activated K⁺ channels. Bicuculline methiodide facilitates burst firing via blocking apamin-sensitive Ca²⁺-activated K⁺ current^[1].

In Vitro

Bicuculline methiodide (30 μM) promotes N-methyl-d-aspartate (NMDA) stimulation to facilitate burst firing in dopamine neurons^[1]. Cluster discharges in the lateral habenular nucleus (LHb) of the antireward center are sufficient conditions for depression to occur. LHb neurons are usually classified into three types: silent, tonic-firing, burst-firing^[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Johnson SW, et al. Bicuculline methiodide potentiates NMDA-dependent burst firing in rat dopamine neurons by blocking apamin-sensitive Ca²⁺-activated K⁺ currents. *Neurosci Lett.* 1997 Aug 1;231(1):13-6.

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

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