

Cinromide

Catalog No: tcsc0013054

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Available Sizes

Size: 100mg

Specifications

CAS No:

58473-74-8

Formula:

 $\mathsf{C}_{11}\mathsf{H}_{12}\mathsf{BrNO}$

Pathway:

Others

Target:

Others

Purity / Grade:

>98%

Solubility:

DMSO : ≥ 310 mg/mL (1219.90 mM)

Alternative Names:

trans-3-Bromo-N-ethylcinnamamide

Observed Molecular Weight:

254.12

Product Description

Cinromide is a broad-spectrum anticonvulsant agent.

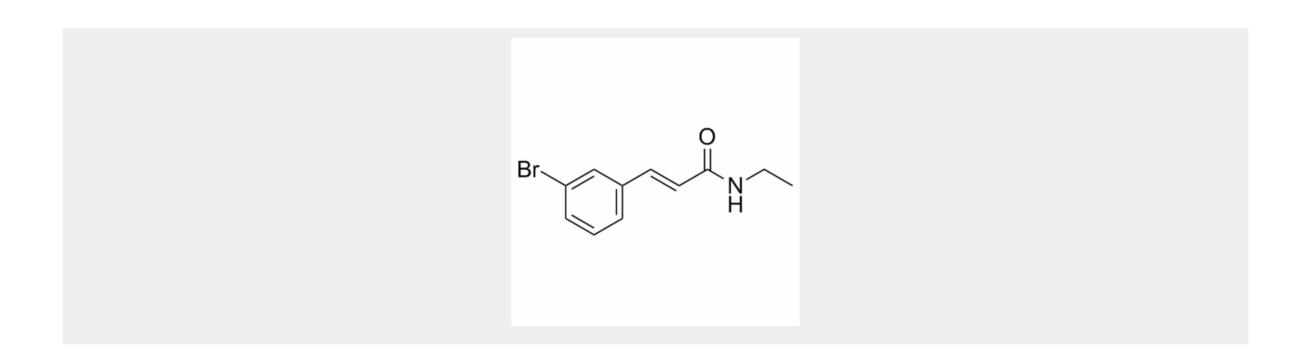
In Vitro: Cinromide (10-100 μ M) inhibits 5-HT-induced contractions in rat fundus strips by 46%. Cinromide (100 μ M) inhibits monoamine oxidase prepared from both liver and brain of rats^[1].

In Vivo:

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Cinromide shows electroshock convulsion and leptazol(pentetrazo1)-induced convulsion in mice, with ED_{50} s of 60 ± 11 mg/kg, 90 ± 15 mg/kg and 80 ± 15 mg/kg, 300 ± 61 mg/kg for i.p. and oral administrion, respectively. Cinromide produces a dose-related antileptazol activity with an ED_{50} value of 58 ± 11 mg/kg by i.p. administration in rats. Furthermore, Cinromide (75 mg/kg) significantly elevates the amount of leptazol needed to induce clonic seizures in the intravenously infused leptazol-threshold test in rats. Cinromide (300 mg/kg, i.p) shows no sifnificant effect on the anaesthetized open-chested dogs after 4 h treatment, neither in conscious dogs after 5-h oral treatment with 300 and 600 mg/kg of Cinromide^[1]. Cinromide (40 mg/kg, i.v.) depresses the response of the neuron to the unconditioned maxillary nerve stimulus, increasing the latency and decreasing the number of spikes, and depresses the response of the neuron to the unconditioned maxillary nerve stimulus, increases the latency and decreasing the latency and decreasing the number of spikes, and depresses the response of the neuron to the unconditioned maxillary nerve stimulus, increasing the latency and decreasing the number of spikes, and depresses the response of the neuron to the unconditioned maxillary nerve stimulus, increasing the latency and decreasing the number of spikes. Cinromide (20, 40, 80 mg/kg, i.v.) increases the latency of the unconditioned response and segmental inhibition dose-dependently. Cinromide decreases periventricular inhibition and $EEG^{[2]}$.



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