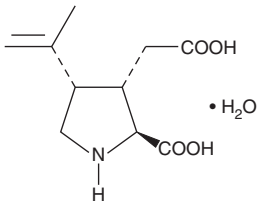
PRODUCT INFORMATION

(−)-(α)-Kainic Acid (hydrate)

Item No. 78050

CAS Registry No.: 58002-62-3
Formal Name: 2S-carboxy-4S-(1-methylethenyl)-3S-pyrrolidineacetic acid, monohydrate
MF: $C_{10}H_{15}NO_4 \bullet H_2O$
FW: 231.2
Purity: ≥98%
Supplied as: A crystalline solid
Storage: -20°C
Stability: ≥4 years



Information represents the product specifications. Batch specific analytical results are provided on each certificate of analysis.

Laboratory Procedures

(−)-(α)-Kainic acid (hydrate) is supplied as a crystalline solid. Aqueous solutions of (−)-(α)-kainic acid (hydrate) can be prepared by directly dissolving the crystalline solid in aqueous buffers. The solubility of (−)-(α)-kainic acid (hydrate) in PBS (pH 7.2) is approximately 50 mg/ml. We do not recommend storing the aqueous solution for more than one day.

Description

(−)-(α)-Kainic acid is a cyclic analog of L-glutamate originally isolated from *D. simplex* that has neuroexcitatory activities.[1] It binds to the homomeric kainate receptors GluK1, GluK2, GluK3, GluK4, and GluK5 (K_is = 75.9, 12.7, 32.8, 4.7, and 15 nM, respectively).[2] (−)-(α)-Kainic acid (5 mM) induces calcium influx and membrane depolarization, as well as glutamate release, in rat brain synaptosomes.[3] It induces chromatin condensation and nuclear membrane fragmentation, markers of apoptosis, in primary neonatal cerebellar granule neurons when used at a concentration of 100 µM.[4] Intracerebroventricular administration of (−)-(α)-kainic acid induces convulsive behavior in rats (ED_{50} = 0.51 nmol/animal) and induces seizures in mice with a 50% convulsive dose (CD_{50}) value of 0.39 nmol/animal.[5] It has been commonly used to induce seizures in rodents.[6,7]

References

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3. Pastuszko, A., Wilson, D.F., and Erecińska, M. Effects of kainic acid in rat brain synaptosomes: The involvement of calcium. *J. Neurochem.* **43(3)**, 747-754 (1984).
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5. Chiamulera, C., Costa, S., Valerio, E., *et al.* Domoic acid toxicity in rats and mice after intracerebroventricular administration: Comparison with excitatory amino acid agonists. *Pharmacol. Toxicol.* **70(2)**, 115-120 (1992).
6. Wang, Q., Yu, S., Simonyi, A., *et al.* Kainic acid-mediated excitotoxicity as a model for neurodegeneration. *Mol. Neurobiol.* **31(1-3)**, 3-16 (2005).
7. Domin, H., Zieba, B., Golembiowska, K., *et al.* Neuroprotective potential of mGluR5 antagonist MTEP: effects on kainate-induced excitotoxicity in the rat hippocampus. *Pharmacol. Rep.* **62(6)**, 1051-1061 (2010).

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