Abecarnil

Cat. No.: HY-105115

CAS No.: 111841-85-1

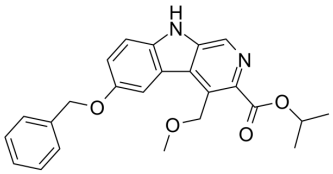
Molecular Formula: $C_{24}H_{24}N_2O_4$

Molecular Weight: 404.46

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

Abecarnil (ZK 112119) is a ligand or a partial agonist for benzodiazepine (BZ) receptor. Abecarnil possesses anxiolytic and anticonvulsant properties. Abecarnil can act as a positive allosteric modulator of $GABA_A$ receptor. Abecarnil inhibits the binding of the BZ [3H]lormetazepam to rat cerebral cortex membranes, with an IC_{50} of 0.82 nM. Abecarnil can be used for epilepsy research[1][2][3][4].

In Vitro

Abecarnil enhances the binding of t-[35S]butylbicyclophosphorothionate to rat cortical membranes[1].
Abecarnil exhibits a 3- to 6-fold higher affinity to forebrain BZ receptors than Diazepam (DZP)[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Abecarnil (0.3 mg/kg, IP, once) antagonizes the brain neuroactive steroid increase induced by foot shock[2].
Abecarnil (0-2.5 mg/kg, IP, once) dose dependently reduces epileptic activity[3].
Abecarnil is effective against sound-induced convulsions in DBA/2 mice, against air blast-induced generalized seizures in gerbils and against myoclonus in baboons Papio papio[4].
Abecarnil is 2-10 times more potent than DZP in most rodent tests of anxiolytic activity, and in reducing locomotor activity in mice and rats thoroughly habituated to the test chamber[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male Sprague-Dawley CD rats (200-250 g)[2]
Dosage:	0.3 mg/kg
Administration:	IP, once, given 30 min before sacrifice
Result:	Failed to change the basal pregnenolone and progesterone, while only slightly decreased THDOC levels, but antagonized the brain neuroactive steroid increase induced by foot shock.

Animal Model:	WAG/Rij rats (male and female, 190-380 g, age 13-19 weeks, 8 rats each group)[3]
Dosage:	0, 0.16, 0.4, 1.0, and 2.5 mg/kg; 1 mL/400 g
Administration:	IP, once

Result:	Reduced the duration of spike-wave discharges and increased immobile behavior. Dose dependently reduced epileptic activity, whether measured as number, mean duration, or total duration of spike-wave discharges. The ED_{50} for reducing the number of spike-wave discharges in the second hour was 0.4 mg/kg.

REFERENCES

[1]. Stephens DN, et al. Abecarnil, a metabolically stable, anxioselective beta-carboline acting at benzodiazepine receptors. J Pharmacol Exp Ther. 1990 Apr;253(1):334-43.

[2]. Barbaccia ML, et al. Stress-induced increase in brain neuroactive steroids: antagonism by abecarnil. Pharmacol Biochem Behav. 1996 May;54(1):205-10.

[3]. Coenen AM, et al. Effects of the beta-carboline abecarnil on epileptic activity, EEG, sleep and behavior of rats. Pharmacol Biochem Behav. 1992 Jul;42(3):401-5.

[4]. Turski L, et al. Anticonvulsant action of the beta-carboline abecarnil: studies in rodents and baboon, Papio papio. J Pharmacol Exp Ther. 1990 Apr;253(1):344-52.

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