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Eglumegad

Cat. No.: HY-18941

CAS No.: 176199-48-7

Molecular Formula: $C_8H_{11}NO_4$

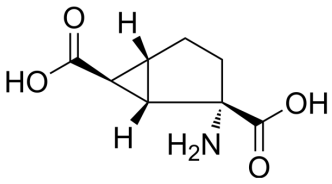
Molecular Weight: 185.18

Target: mGluR

Pathway: GPCR/G Protein; Neuronal Signaling

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : 33.33 mg/mL (179.99 mM; ultrasonic and warming and heat to 60°C)

H_2O : 6.33 mg/mL (34.18 mM; Need ultrasonic and warming)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	5.4002 mL	27.0008 mL	54.0015 mL
	5 mM	1.0800 mL	5.4002 mL	10.8003 mL
	10 mM	0.5400 mL	2.7001 mL	5.4002 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 2.86 mg/mL (15.44 mM); Clear solution; Need ultrasonic and warming and heat to 60°C
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (13.50 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Eglumegad (LY354740) is a highly potent and selective group II (mGlu2/3) receptor agonist with IC_{50}s of 5 nM and 24 nM on transfected human mGlu2 and mGlu3 receptors, respectively.

IC_{50} & Target

mGluR2R	mGluR3R
5 nM (IC_{50})	24 nM (IC_{50})

In Vitro

Eglumegad (LY354740) down-regulates spots 1014, 1822 (hypoxia up-regulated protein 1), 4513 (an isoform of protein disulfide isomerase 3), 6204, 6312, 7306 (26S proteasome non-ATPase regulatory subunit 7) and protein spots 1013 and 6005 (destrin), and up-regulates spot 6507 (collapsin response mediator protein 1) in mouse cortical neurons[2].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

In Vivo

Eglumegad (LY354740) (15 or 30 mg/kg, i.p.) has no effect on spatial working memory performance in Gria1 $^{/}$ or WT mice, and it has no effect on rewarded alternation testing with a short inter-trial interval in Gria1 $^{/}$ and WT mice at concentration of 30 mg/kg. Eglumegad (LY354740) (15 or 30 mg/kg, i.p.) reduces spontaneous locomotor activity in wild-type and Gria1 $^{/}$ mice[1]. Eglumegad (LY354740) (15 mg/kg, i.p.) dreases novelty-induced hyperlocomotion in naive GluA1-KO and pre-handled GluA1-KO males, but not in females. Eglumegad (LY354740) (15 mg/kg, i.p.) significantly reduces the increased c-Fos expression of GluA1-KO males to the level of WT males, but not in of females[3]. Eglumegad (LY354740) (10 mg/kg, i.p.) attenuates the immobilization stress-induced increase in BDNF mRNA expression in the rat mPFC[4].

MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1][4]

Mice[1]

In Experiment 1A wild-type (female: N=6; male: N=5) and Gria1$^{-/-}$ mice (female: N=7; male: N=8) first receive 30 trials of drug-free testing (five trials per day for 6 days). Each animal is then tested on rewarded alternation following an injection of either Eglumegad (LY354740) (15 mg/kg) or vehicle. After injection animals are returned to the home cage for 30 min before behavioural testing commenced. Each animal is given 10 trials of rewarded alternation in the T-maze. Mice are given a maximum of 120 s to a complete a trial. At least 24 h after the first round of drug testing the animals are re-tested in the absence of any drug treatment to ensure that there are no long-term effects of the drug, and that the mice maintain a high level of alternation performance. Twenty-four hours after this re-testing, mice receive a further 10 trials of rewarded alternation testing, but now under the drug condition that they have not previously received. The order of drug exposure is counterbalanced within genotype and sex as far as possible given the numbers of mice. In all stages, the number of trials in which the animal alternated, as well as time taken to run from the start arm to the food well on the sample run (sample latency), and the time taken to run from the start arm to making a choice on the choice run (choice latency), are recorded. Latencies are measured by the experimenter using a stopwatch. The experimenter is blind to the genotype and drug allocations of the animals throughout testing. In Experiment 1B, separate groups of male wild-type (N=7) and Gria1$^{-/-}$ mice (N=7) undgo the same procedure as Experiment 1A but now they receive either vehicle or a higher dose of Eglumegad (LY354740) (30 mg/kg). Subsequently, in Experiment 1C, to investigate the potential effects of increased proactive interference, the procedure used in Experiment 1B is repeated in the same mice, using the same drug dose (30 mg/kg), but now using a modified testing protocol in which the interval between trials is reduced to 20 s.

Rats[4]

Two experiments are conducted. The first experiment compares the effects of LY354740 (10 mg/kg, i.p., neutralized to a pH ~ 7.4) or vehicle (0.9% saline neutralized to pH ~ 7.4) in rats remaining in their home cages or rats exposed to 2 h of immobilization stress in plastic cones (n=7, cage control/vehicle; n=7, cage control/LY354740; n=6, stress/vehicle; n=6, stress/Eglumegad (LY354740)). The second experiment compares two lower LY354740 doses (1 and 3 mg/kg, i.p.) or vehicle in rats exposed to a 2-h period of immobilization stress to rats treated with vehicle in their home cages (n=4 for all groups). All animals are injected with either Eglumegad (LY354740) or vehicle 15 min prior to being placed in plastic cones with the open end securely closed. All immobilized rats are placed in plexiglass chamber with animal bedding on the bottom; brought to a quiet room outside of the animal colony; and immediately placed in a plastic cone. Two hours after being placed in the plastic containers, the rats are decapitated. The brains are removed and frozen on dry ice, and stored at −80°C.

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CUSTOMER VALIDATION

- Neurobiol Dis. 2020 Jun;139:104807.
- Front Pharmacol. 2020 Feb 28;11:183.
- Pharmacol Biochem Behav. 2023 Sep 14;231:173637.

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REFERENCES

[1]. Boerner T, et al. The group II metabotropic glutamate receptor agonist LY354740 and the D2 receptor antagonist haloperidol reduce locomotor hyperactivity but fail to rescue spatial working memory in GluA1 knockout mice. Eur J Neurosci. 2017 Apr;45(7):912-

[2]. Orlando R, et al. Levels of the Rab GDP dissociation inhibitor (GDI) are altered in the prenatal restrain stress mouse model of schizophrenia and are differentially regulated by the mGlu2/3 receptor agonists, LY379268 and LY354740. Neuropharmacology. 2014

[3]. Procaccini C, et al. Reversal of novelty-induced hyperlocomotion and hippocampal c-Fos expression in GluA1 knockout male mice by the mGluR2/3 agonist LY354740. Neuroscience. 2013 Oct 10;250:189-200

[4]. Lee Y, et al. The mGlu2/3 receptor agonist LY354740 suppresses immobilization stress-induced increase in rat prefrontal cortical BDNF mRNA expression. Neurosci Lett. 2006 May 8;398(3):328-32.

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