LM22A-4

Cat. No.: HY-100673

CAS No.: 37988-18-4

Molecular Formula: $C_{15}H_{21}N_3O_6$

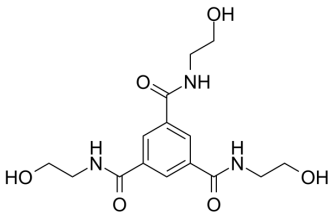
Molecular Weight: 339.34

Target: Trk Receptor

Pathway: Neuronal Signaling; Protein Tyrosine Kinase/RTK

Storage:

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



SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 50 mg/mL (147.34 mM)

DMSO : ≥ 29 mg/mL (85.46 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.9469 mL	14.7345 mL	29.4690 mL
5 mM	0.5894 mL	2.9469 mL	5.8938 mL
10 mM	0.2947 mL	1.4734 mL	2.9469 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 120 mg/mL (353.63 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (7.37 mM); Clear solution

BIOLOGICAL ACTIVITY

Description LM22A-4 is a specific agonist of tyrosine kinase receptor B, used for neurological disease research.

In Vitro LM22A-4 significantly up-regulates OPN and ALPase mRNA expression in a dose-dependent manner and OC mRNA level is

	significantly increased by 5 μM of LM22A-4. LM22A-4 significantly increases OPN, ALPase and OC mRNA expression in a time-dependent manner. LM22A-4 stimulated OPN and OC mRNA expression in HCEM cells cultured with mineralizing media[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	LM22A-4 (10 mg/kg, i.p.) significantly reduces the degree of tissue injury and apoptosis (TUNEL staining and caspase-3 and Bcl-2 expression) compared with vehicle treated group. LM22A-4 also significantly ameliorates the recovery of limb function. LM22A-4 (10 mg/kg) treatment results in a significant increase in neuron number. LM22A-4 administration (10 mg/kg) significantly improves the neurological scores compared with those of the solvent-treated animals[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Animal Administration [1]	ICR mice are randomly divided into five groups: sham treatment, spinal cord injury, spinal cord injury combined with solvent treatment, spinal cord injury combined with LM22A-4 treatment (10 mg/kg), and spinal cord injury combined with LM22A-4 treatment (15 mg/kg), with each group containing 26 animals. Preparation of the mouse SCI model is based on a previous study and on a protocol employed by this group. Briefly, a mouse is anesthetized via the administration of chloral hydrate (4 mg/kg) before a 3-cm incision is introduced on its back. T7-T11 vertebrae are exposed under a surgical microscope before the laminae are removed with a vascular clip to fully expose the spinal cord. The spinal cord is clamped with the vascular clip for 1 minute with a force of 10 g. The animal is then subjected to complete staunching of the bleeding, and the incised dorsal muscle and skin are sutured. Mice from the control group undergo laminectomy, full exposure of the spinal cord, and subsequent suturing, but without aortic clamping. After the surgery, the mice are placed on a warm blanket until fully awake and are then housed in cages accommodating a normal diet. After the SCI treatment, 14 mice are sacrificed to enable molecular and histological examinations; the other 14 mice are allowed to live for 20 days for neurological scoring and are sacrificed on day 20 via cervical dislocation. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

REFERENCES

[1]. Yu G, et al. Protective effects of LM22A-4 on injured spinal cord nerves. Int J Clin Exp Pathol. 2015 Jun 1;8(6):6526-32. eCollection 2015.

[2]. Kajiya M, et al. BDNF mimetic compound LM22A-4 regulates cementoblast differentiation via the TrkB-ERK/Akt signaling cascade. Int Immunopharmacol. 2014 Apr;19(2):245-52

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