Trofinetide

Cat. No.: HY-16757

CAS No.: 853400-76-7

Molecular Formula: $C_{13}H_{21}N_3O_6$

Molecular Weight: 315.32

Target: Others

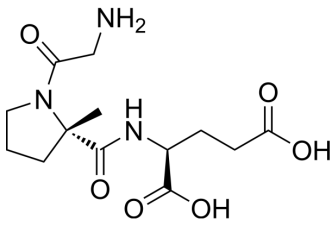
Pathway: Others

Storage: Sealed storage, away from moisture and light, under nitrogen

Powder -80°C 2 years

-20°C 1 year

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture and light, under nitrogen)



SOLVENT & SOLUBILITY

In Vitro

H_2O : 110 mg/mL (348.85 mM; Need ultrasonic)

H_2O : ≥ 50 mg/mL (158.57 mM)

DMSO : 25 mg/mL (79.28 mM; Need ultrasonic)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	3.1714 mL	15.8569 mL	31.7138 mL
	5 mM	0.6343 mL	3.1714 mL	6.3428 mL
	10 mM	0.3171 mL	1.5857 mL	3.1714 mL

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

In Vivo

1. Add each solvent one by one: PBS

Solubility: 100 mg/mL (317.14 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Trofinetide (NNZ-2566), a synthetic analogue of the endogenous N-terminus tripeptide, Glycine-Proline-Glutamate (GPE), has been shown to be neuroprotective in animal models of brain injury.

In Vivo

Trofinetide (NNZ-2566) suppresses penetrating ballistic-like brain injury (PBBI) induced inflammatory cell infiltration at 3 days following PBBI as compare to vehicle treatment. Trofinetide treatment significantly reduces the elevation of IL-6 (79%), E-selectin (81%), IL-1β (76%) and TNF-α (72%) mRNA levels in the injured hemisphere at 12 h post-PBBI, with maximal inhibition occurring between 12 h and 24 h. Trofinetide treatment does not affect the PBBI-induced up-regulation of IL-6 expression at any time point, but does produce significant reductions in the injury-induced up-regulation of IL-1β, INF-γ, and

TNF-α expression. Trofinetide treatment suppresses IL-1β expression in the injured brain hemisphere for up to 7 days post-PBBI[1]. The high doses of Trofinetide (NNZ-2566) (10 and 100 mg/kg bolus followed by continuous infusion) attenuate non-convulsive seizure (NCS) occurring beyond 2 h after permanent middle cerebral artery occlusion (pMCAo). All doses of Trofinetide completely suppress the delayed occurrence of NCS as compare with the vehicle-treated animals[2].

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

PROTOCOL

Animal Administration [1]

Three groups of eight rats are evaluated: vehicle/sham, vehicle/penetrating ballistic-like brain injury (PBBI), Trofinetide (NNZ-2566)/PBBI. A bolus injection of 10 mg/kg Trofinetide or 1 mL/kg saline (vehicle) is administered intravenously (IV) to each animal at 30 minutes post-PBBI surgery, immediately followed by a continuous IV infusion of Trofinetide at a rate of 3 mg/kg/h or an equal volume of vehicle for various durations (1 h, 4 h, or 12 h). Rats are subsequently euthanized and brain tissues are collected for processing at 1 h, 4 h, 12 h, 24 h, 3, and 7 days following the initiation of treatment[1].

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

REFERENCES

[1]. Wei HH, et al. NNZ-2566 treatment inhibits neuroinflammation and pro-inflammatory cytokine expression induced by experimental penetrating ballistic-like brain injury in rats. J Neuroinflammation. 2009 Aug 5;6:19.

[2]. Lu XC, et al. NNZ-2566, a glypromate analog, attenuates brain ischemia-induced non-convulsive seizures in rats. J Cereb Blood Flow Metab. 2009 Dec;29(12):1924-32.

[3]. Cartagena CM, Phillips KL, Williams GL, et al. Mechanism of action for NNZ-2566 anti-inflammatory effects following PBBI involves upregulation of immunomodulator ATF3. Neuromolecular Med. 2013;15(3):504-514.

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