LQVTDSGLYRCVIYHPP TFA

Cat. No.: HY-P3400A

Molecular Formula: $C_{91}H_{138}F_3N_{23}O_{27}S$

Molecular Weight: 2075.27

Sequence Shortening: LQVTDSGLYRCVIYHPP

Target: Others

Pathway: Others

Storage: Sealed storage, away from moisture and light
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)

LQVTDSGLYRCVIYHPP (TFA salt)

SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (48.19 mM)
H_2O : < 0.1 mg/mL (ultrasonic;adjust pH to 3 with HCl) (insoluble)
* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	0.4819 mL	2.4093 mL	4.8187 mL
	5 mM	0.0964 mL	0.4819 mL	0.9637 mL
	10 mM	0.0482 mL	0.2409 mL	0.4819 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (1.20 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 2.5 mg/mL (1.20 mM); Suspended solution; Need ultrasonic
3. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (1.20 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

LQVTDSGLYRCVIYHPP (LP17) TFA is a triggering receptor expressed on myeloid cells (TREM-1) inhibitory peptide. LQVTDSGLYRCVIYHPP TFA substantially alleviates ischemia-induced infarction and neuronal injury. LQVTDSGLYRCVIYHPP TFA can get access into brain and block TREM-1[1].

IC_{50} & Target	TREM-1[1]

In Vitro

LQVTDSGLYRCVIYHPP (LP17) (1 or 10 μM; 24 h) substantially decreases mRNA levels of pro-inflammatory cytokines and chemokines after reoxygenation and remarkably attenuates extracellular protein levels of IL-1β and IL-18 in a microglia oxygen-glucose deprivation (OGD) model[1].
LQVTDSGLYRCVIYHPP (LP17) (10 μM; 24 h) interacts with microglial SYK[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

RT-PCR[1]

Cell Line:	Primary microglia
Concentration:	1 or 10 μM
Incubation Time:	24 h
Result:	Decreased mRNA levels of NLRP3, IL-1β, IL-18, IL-6, CD16, CD32, iNOS, MCP-1, CXCL-1, and CXCL-2 after reoxygenation.

Western Blot Analysis[1]

Cell Line:	Primary microglia
Concentration:	10 μM
Incubation Time:	24 h
Result:	Suppressed ischemia/reperfusion-induced increments in CARD9, p-p65 in CARD9/NF-κB signaling and NLRP3, ASC, cleaved caspase-1, mature IL-1β, and mature IL-18 in NLRP3/caspase-1 signaling in a microglia oxygen-glucose deprivation (OGD) model.

In Vivo

LQVTDSGLYRCVIYHPP (LP17) (0.5 or 1 mg/kg; intranasal; daily for 3 days) alleviates ischemia-induced infarction and neuronal injury in mice[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Adult male C57BL/6J mice (20-25 g), mice cerebral ischemia/reperfusion (I/R) model induced by middle cerebral artery occlusion (MCAO)[1]
Dosage:	0.5 mg/kg or 1 mg/kg
Administration:	Intranasal administration, once daily for 3 consecutive days after MCAO
Result:	Abolished ischemia-induced TREM-1 elevation at 1 mg/kg. Significantly reduced infarct volume by 27.3%, induced a markedly reduction in TUNEL positive cells and FJC positive neurons at 1 mg/kg. Rescued neurological deficits and cognitive dysfunction of MCAO mice. Inhibited microglial M1 polarization and neutrophil infiltration.

REFERENCES

[1]. Pengfei Xu, et al. Microglial TREM-1 receptor mediates neuroinflammatory injury via interaction with SYK in experimental ischemic stroke. Cell Death Dis. 2019 Jul 19;10(8):555.

Caution: Product has not been fully validated for medical applications. For research use only.

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