LQVT	DSGL	YRCVI	YHPP
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Cat. No.:	НҮ-Р3400	
CAS No.:	887255-16-5	
Molecular Formula:	C ₈₉ H ₁₃₇ N ₂₃ O ₂₅ S	
Molecular Weight:	1961.24	
Sequence Shortening:	LQVTDSGLYRCVIYHPP	
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

		Solvent Mass Concentration	1 mg	5 mg	10 mg	
	Preparing Stock Solutions	1 mM	0.5099 mL	2.5494 mL	5.0988 mL	
		5 mM	0.1020 mL	0.5099 mL	1.0198 mL	
		10 mM	0.0510 mL	0.2549 mL	0.5099 mL	

BIOLOGICAL ACTIV	
Description	LQVTDSGLYRCVIYHPP (LP17) is a triggering receptor expressed on myeloid cells (TREM-1) inhibitory peptide. LQVTDSGLYRCVIYHPP substantially alleviates ischemia-induced infarction and neuronal injury. LQVTDSGLYRCVIYHPP can get access into brain and block TREM-1 ^[1] .
IC₅₀ & 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 μΜ
	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.
/ivo	neuronal injury in mice	LP17) (0.5 or 1 mg/kg; intranasal; daily for 3 days) alleviates ischemia-induced infarction and ^[1] . ently 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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