IKKγ NBD Inhibitory Peptide TFA

Cat. No.: HY-P1847A

Molecular Formula: $C_{172}H_{260}N_{49}F_3O_{44}S_1$

Molecular Weight: 3807.32

Sequence Shortening: DRQIKIWFQNRRMKWKKTALDWSWLQTE

Target: NF-κB

Pathway: NF-κB

Storage: Sealed storage, away from moisture

Powder	-80°C	2 years
	-20°C	1 year

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

BIOLOGICAL ACTIVITY

Description

IKKγ NBD Inhibitory Peptide TFA is a highly specific inhibitor NF-κB inhibitor. IKKγ NBD Inhibitory Peptide TFA acts by disrupting the interaction between IKKγ/NEMO-binding domain (NBD) with IKKα and IKKβ, thus blocking TNF-α-induced NF-kB activation. IKKγ NBD Inhibitory Peptide TFA could significantly suppresses inflammation and ameliorate the cerebral ischemia-induced neurological deficits[1][2][3].

In Vitro

In a canonical pathway, NF kappa B activation depends on the IKK complex activity, which is formed by three subunits (IKKκ and IKKβ and IKKγ/NEMO), thus the KKγ NBD Inhibitory Peptide TFA inhibits TNF-α-induced NF-kB activation[2].

IKKγ NBD Inhibitory Peptide TFA (10 μM; 90 min) prevents Doxorubicin (HY-15142A)-induced (15 μM; 4 h) p65 phosphorylation in BT-474 cells[2].

IKKγ NBD Inhibitory Peptide TFA (10 μM; 48 h) slightly inhibits BT-474 cell viability while it markedly enhances the effects on Doxorubicin[2].

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

In Vivo

IKKγ NBD Inhibitory Peptide TFA (40 μg/4 μL; ICV; single dose; 2 h before MCAO) exhibits neuroprotective effect with reduction in DNA fragmentation, and its ischemic brain damage reduction mechanism be attributed to reduction in inflammation following ischemic injury in middle cerebral artery occlusion (MCAO) rats model[3].

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Animal Model:	Adult male Sprague-Dawley rats (255 g) with middle cerebral artery occlusion (MCAO)[3]
Dosage:	40 μg/4 μL
Administration:	Intracerebroventricular injection; 2 h before MCAO induction
Result:	Significantly reduced striatal IL-1b level in IKK-NBD peptide treated rats. Also resulted in reduced staining of microglial OX-42 and protection of BBB from ischemic insult.

REFERENCES

[1]. Tapia MA, et al. Inhibition of the canonical IKK/NF kappa B pathway sensitizes human cancer cells to doxorubicin. Cell Cycle. 2007 Sep 15;6(18):2284-92.

[2]. Desai A, et al. Neuroprotective potential of the NF-κB inhibitor peptide IKK-NBD in cerebral ischemia-reperfusion injury. Neurochem Int. 2010 Dec;57(8):876-83.

[3]. Zhao J, et al. Development of novel NEMO-binding domain mimetics for inhibiting IKK/NF-κB activation. PLoS Biol. 2018 Jun 11;16(6):e2004663.

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