Dynorphin A TFA

Cat. No.:	HY-P1333A
Molecular Formula:	$C_{101}H_{156}F_3N_{31}O_{25}$
Molecular Weight:	2261.5
Sequence:	Tyr-Gly-Gly-Phe-Leu-Arg-Arg-Ile-Arg-Pro-Lys-Leu-Lys-Trp-Asp-Asn-Gln
Sequence Shortening:	YGGFLRRIRPKLKWDNQ
Target:	Opioid Receptor; Endogenous Metabolite; Apoptosis; Caspase
Pathway:	GPCR/G Protein; Neuronal Signaling; Metabolic Enzyme/Protease; Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description

Dynorphin A TFA is an endogenous opioid peptide involved in inhibitory neurotransmission in the central nervous system (CNS). Dynorphin A TFA is a highy potent kappa opioid receptor (KOR) agonist, and is also an agonist for other opioid receptors, such as mu (MOR) and delta (DOR). Dynorphin A TFA can induce neuronal death, and can be used in the research of neurological disease[1][2].

IC_{50} & Target		
Human Endogenous Metabolite	Caspase-3	κ Opioid Receptor/KOR

In Vitro

Dynorphin A TFA (10 μM, 4 h/72 h) increases caspase-3 activity and the level of cytochrome c released from mitochondria in mouse striatal neurons, and induces neuronal death[3].
dynorphin A TFA (33 μM, 4 h) elevates $[Ca^{2+}]_i$ and causes a significant loss of neurons[4].
dynorphin A TFA (1 μM) inhibits the release of vasopressin (VP) from the isolated neural lobe[5].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[3]

Cell Line:	Mouse striatal neurons
Concentration:	10 μM
Incubation Time:	0, 24, 48, 72 h
Result:	Induced neuronal death (identified by the fragmentation and destruction of the cell body and neurites).

In Vivo

Dynorphin A TFA (intracerebroventricular injection, 1 μg of 2 μL, a single dose) inhibits vasopressin (VP) release in 24 h water-deprived male rats[5].
Dynorphin A TFA (intracerebroventricular injection, 500 pmol/5 μL per day for 4 d) alleviates stress-induced behavioral impairments in ddY mice accompanied by regulation of the 5-HTergic system in the brain[6].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	24 h water-deprived male rats[5]

Dosage:	1 μg of 2 μL
Administration:	Intracerebroventricular injection
Result:	Inhibited vasopressin (VP) release 30 min upon injection.

Animal Model:	Male ddY mice[6]
Dosage:	15, 150, 1500 pmol/5 μL per day for 4 days
Administration:	Intracerebroventricular injection
Result:	Attenuated the repeated stress-induced escape failures from the shock.

REFERENCES

[1]. Aruna Sharma, et al. Monoclonal antibodies as novel neurotherapeutic agents in CNS injury and repair. Int Rev Neurobiol. 2012;102:23-45.

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[3]. B J Van de Heijning, et al. Dynorphin-A and vasopressin release in the rat: a structure-activity study. Neuropeptides. 1994 Jun;26(6):371-8.

[4]. Takayoshi Mamiya, et al. Dynorphin a (1-13) alleviated stress-induced behavioral impairments in mice. Biol Pharm Bull. 2014;37(8):1269-73.

[5]. K F Hauser, et al. Dynorphin A (1-13) neurotoxicity in vitro: opioid and non-opioid mechanisms in mouse spinal cord neurons. Exp Neurol. 1999 Dec;160(2):361-75.

[6]. Zhang, et al.Dynorphin A as a Potential Endogenous Ligand for Four Members of the Opioid Receptor Gene Family. J Pharmacol Exp Ther. 1998 Jul;286(1):136-41.

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