TAT-DEF-Elk-1 TFA

Cat. No.:	HY-P2262A
Molecular Formula:	$C_{157}H_{260}N_{57}F_3O_{42}$
Molecular Weight:	3675.09
Sequence:	Gly-Arg-Lys-Lys-Arg-Arg-Gln-Arg-Arg-Arg-Pro-Pro-Ser-Pro-Ala-Lys-Leu-Ser-Phe-Gln-Phe-Pro-Ser-Ser-Gly-Ser-Ala-Gln-Val-His-Ile
Sequence Shortening:	GRKKRRQRRRPPSPAKLSFQFPSSGSAQVHI
Target:	Others
Pathway:	Others
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	TAT-DEF-Elk-1 TFA (TDE TFA) is a cell-penetrating peptide inhibitor of Elk-1, mimics and specifically interferes with the DEF domain of Elk-1. TAT-DEF-Elk-1 TFA blocks Elk-1 phosphorylation and prevents Elk-1 nuclear translocation without interfering with ERK nor MSK1 activation. TAT-DEF-Elk-1 TFA is a useful tool to analyze the role of Elk-1 in this process during the development of neuronal plasticity[1].
IC$_{50}$ & Target	IC50: Elk-1[1]
In Vitro	Elk-1 phosphorylation on Ser383/389 has a dual function and triggers both Elk-1 nuclear translocation and SRE-dependent gene expression[1]. TAT-DEF-Elk-1 TFA (5 µM; 1 hour) specifically inhibits glutamate-induced elk-1 activation and does not interfer with ERK, MSK-1, or CREB phosphorylation[1]. TAT-DEF-Elk-1 TFA (5-10 µM; 2 hour) treatment shows a significant inhibition of c-Fos, Zif268 and JunB, but has no effects on c-Jun expression[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only. Western Blot Analysis[2]

Cell Line:	Neurons
Concentration:	5 µM; 10 µM
Incubation Time:	1 hour
Result:	Decreased elk-1 expression and had no effects on ERK, MSK-1, or CREB phosphorylation.

RT-PCR[2]

Cell Line:	Primary striatal neurons
Concentration:	5 µM

	Incubation Time:	2 hour
	Result:	Decreased c-Fos, Zif268 and JunB mRNA level but did not effect c-Jun.
In Vivo	TAT-DEF-Elk-1 TFA (intraperitoneal injection; 1mg/kg; daily; 14 days) reflects antidepressant efficacy in mice, it decreases immobility similar to the reference antidepressants fluoxetine and desipramine (DMI)[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.	
	Animal Model:	C57Bl6 mice (3-6 months old males) are subjected to social defeat stress[2]
	Dosage:	1 mg/kg
	Administration:	Intraperitoneal injection; daily; 14 days
	Result:	Reversed social-defeat induced decrease of hippocampal Bdnf expression by repeated TDE administration.

CUSTOMER VALIDATION

- Research Square Preprint. 2022 Jan.

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REFERENCES

[1]. Lavaur J, et al. A TAT-DEF-Elk-1 peptide regulates the cytonuclear trafficking of Elk-1 and controls cytoskeleton dynamics.J Neurosci. 2007 Dec 26;27(52):14448-58.

[2]. Apazoglou K, et al. Antidepressive effects of targeting ELK-1 signal transduction.Nat Med. 2018 May;24(5):591-597.

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

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