TAT-GluN2BCTM

Cat. No.:	HY-P5277
CAS No.:	1587742-50-4
Molecular Formula:	$C_{224}H_{374}N_{86}O_{54}$
Molecular Weight:	5135.91
Sequence:	Tyr-Gly-Arg-Lys-Lys-Arg-Arg-Gln-Arg-Arg-Arg-Lys-Lys-Asn-Arg-Asn-Lys-Leu-Arg-Arg-Gln-His-Ser-Tyr-Lys-Phe-Glu-Arg-Gln-Lys-Ile-Leu-Asp-Gln-Arg-Phe-Phe-Glu
Sequence Shortening:	YGRKKRRQRRRKKNRNKLRRQHSYKFERQKILDQRFFE
Target:	DAPK
Pathway:	Apoptosis
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.

BIOLOGICAL ACTIVITY

Description	TAT-GluN2BCTM is a membrane-permeable DAPK1-targeting peptide. TAT-GluN2BCTM targets active DAPK1 to lysosomes for degradation. TAT-GluN2BCTM protects neurons from oxidative stress and NMDAR-mediated excitotoxicity by knocking down DAPK1. TAT-GluN2BCTM can be used in the study of neuroprotection[1].
IC_{50} & Target	DAPK1[1].

In Vitro

TAT-GluN2BCTM (200 μM; 0.5, 2, 4 h) results in a time-dependent reduction in DAPK1 levels in NMDA-treated primary neuronal cultures [1].

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

Western Blot Analysis[1]

Cell Line:	primary neuronal cultures (NMDA-treated)
Concentration:	200 μM
Incubation Time:	0.5, 2, 4 h
Result:	Reduced DAPK1 levels in a time-dependent manner.

In Vivo

TAT-GluN2BCTM (10 mg/kg; i.v.; single) results in a significant reduction in DAPK1 levels only in the ischemic side of the brain in rats[1].

TAT-GluN2BCTM (10 mg/kg; i.v.; single) shows prominent neuroprotection in the cortex and striatum of rats[1].

TAT-GluN2BCTM is capable of specific knockdown of the active (but not inactive) form of DAPK1[1].

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

Animal Model:	Focal ischemia rat model[1].
Dosage:	10 mg/kg
Administration:	Intravenous injection; single.

Result:	Reduced the levels of DAPK1 only in the ischemic side of the brain. Significantly reduced the numbers of degenerating neurons in both striatum and cortical areas.

REFERENCES

[1]. Fan X, et al. Rapid and reversible knockdown of endogenous proteins by peptide-directed lysosomal degradation. Nat Neurosci. 2014 Mar;17(3):471-80.

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