Tat-NR2B9c

Cat. No.:	HY-P0117
CAS No.:	500992-11-0
Molecular Formula:	$C_{105}H_{188}N_{42}O_{30}$
Molecular Weight:	2518.88
Sequence Shortening:	YGRKKRRQRRRKLSSIESDV
Target:	iGluR; NO Synthase
Pathway:	Membrane Transporter/Ion Channel; Neuronal Signaling; Immunology/Inflammation
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)

YGRKKRRQRRRKLSSIESDV

SOLVENT & SOLUBILITY

In Vitro

H_2O : 100 mg/mL (39.70 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	0.3970 mL	1.9850 mL	3.9700 mL
	5 mM	0.0794 mL	0.3970 mL	0.7940 mL
	10 mM	0.0397 mL	0.1985 mL	0.3970 mL

Please refer to the solubility information to select the appropriate solvent.

In Vivo

1. Tat-NR2B9c is prepared in saline[4].

BIOLOGICAL ACTIVITY

Description

Tat-NR2B9c (Tat-NR2Bct; NA-1) is a postsynaptic density-95 (PSD-95) inhibitor, with EC_{50} values of 6.7 nM and 670 nM for PSD-95d2 (PSD-95 PDZ domain 2) and PSD-95d1, respectively. Tat-NR2B9c disrupts the PSD-95/NMDAR interaction, inhibiting NR2A and NR2B binding to PSD-95 with IC_{50} values of 0.5 μM and 8 μM, respectively. Tat-NR2B9c also inhibits neuronal nitric oxide synthase (nNOS)/PSD-95 interaction, and possesses neuroprotective efficacy[1][2][5].

IC_{50} & Target		
	NMDA Receptor	nNOS

In Vitro

Tat-NR2B9c is a PSD-95 inhibitor, with an EC_{50} of 6.7 nM for PSD-95d2, representing a >100-fold higher affinity for this domain than for PSD-95d1 (EC_{50}, 0.67 μM). Tat-NR2B9c inhibits NMDAR2A, NMDAR2B, and NMDAR2C binding to PSD-95, with IC_{50}s of 0.5 μM, ~8 μM, and 0.75 μM, respectively.
Tat-NR2B9c also blocks the interaction between PSD-95 and nNOS with an IC_{50} of ~0.2 μM[1].
Tat-NR2B9c reduces association of PSD-95 with GluN2B by ~50% in the YAC128 striatum, decreases NMDA-induced p38

	activation in YAC128 striatal tissue, but shows no effect on the NMDA-induced JNK activation[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Tat-NR2B9c (10 nmol/g, i.v.) reduces infarction volume of male C57BL/6 mice, but has no effect at 3 nM/g[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Postnatal mono-cultured WT and YAC128 striatal neurons (DIV 9, due to the viability of these mono-cultured MSNs) are pretreated for 1 h with 200 nM Tat-NR2B9c, and/or SB-239063 (p38 inhibitor), and/or SP-600125 (JNK inhibitor), then incubated with or without 500 μM NMDA for 10 min. After NMDA treatment, striatal neurons are washed once with warm plating medium (PM) and then incubated in conditioned PM (without Tat peptides or p38, JNK inhibitors) for 24 h. Then cells are washed with PBS once and fixed with 4% paraformaldehyde (PFA) for 30 min[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [3]	Mice[3] In each study mice are randomly allocated to three treatment groups (0.0, 3.0, 10.0 nMole/g Tat-NR2B9c) or to sham treatment. The individual performing the experimental procedures, administering treatments and performing the analyses is blinded to the treatment assignments. Tat-NR2B9c is prepared at the indicated doses and administered intravenously via the tail vein using a pump in a volume of 1 μL/g over 5 min beginning at reperfusion[3]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- J Cereb Blood Flow Metab. 2019 Aug;39(8):1588-1601.
- Neuropharmacology. 2024 Mar 21:109905.
- Sci Rep. 2018 May 18;8(1):7848.
- PLoS One. 2020 Mar 3;15(3):e0229499.
- J Neuropathol Exp Neurol. 2020 Jul 1;79(7):800-808.

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REFERENCES

[1]. Cui H, et al. PDZ protein interactions underlying NMDA receptor-mediated excitotoxicity and neuroprotection by PSD-95 inhibitors. J Neurosci. 2007 Sep 12;27(37):9901-15.

[2]. Fan J, et al. P38 MAPK is involved in enhanced NMDA receptor-dependent excitotoxicity in YAC transgenic mouse model of Huntington disease. Neurobiol Dis. 2012 Mar;45(3):999-1009.

[3]. Teves LM, et al. Efficacy of the PSD95 inhibitor Tat-NR2B9c in mice requires dose translation between species. J Cereb Blood Flow Metab. 2016 Mar;36(3):555-61.

[4]. Jing Fan, et al. N-methyl-D-aspartate Receptor Subunit- And Neuronal-Type Dependence of Excitotoxic Signaling Through Post-Synaptic Density 9. J Neurochem. 2010 Nov;115(4):1045-56.

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

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