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产品名称：Tat-NR2B9c
产品别名： NA-1

生物活性：

Description	Tat-NR2B9c (NA-1) is a 20-aa peptide, which acts as a postsynaptic density-95 (PSD-95) inhibitor, with an EC_{50} of 6.7 nM for PSD-95d2 (PSD-95 PDZ domain 2), and 670 nM for PSD-95d1. Tat-NR2B9c also reduces NMDA-induced p38 activation, inhibits neuronal nitric oxide synthase (nNOS)/PSD-95 interaction, and possesses neuroprotective efficacy.
IC_{50} & Target	EC50: 6.7 nM (PSD-95d2), 670 nM (PSD-95d1)[1] NO synthase[1]
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].
In Vivo	Tat-NR2B9c (10 nM/g, i.v.) reduces infarction volume of male C57BL/6 mice, but has no effect at 3 nM/g[3].
Solvent&Solubility	***In Vitro:*** **H_2O : ≥ 50 mg/mL (19.85 mM)** * "≥" means soluble, but saturation unknown.

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

*请根据产品在不同溶剂中的溶解度选择合适的溶剂配制储备液；一旦配成溶液，请分装保存，避免反复冻融造成的产品失效。

储备液的保存方式和期限：-80°C, 6 months; -20°C, 1 month。-80°C 储存时，请在 6 个月内使用，-20°C 储存时，请在 1 个月内使用。

In Vivo:

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

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]. Huang LE, et al. N-methyl D-aspartate receptor subtype 2B antagonist, Ro 25-6981, attenuates neuropathic pain by inhibiting postsynaptic density 95 expression. Sci Rep. 2018 May 18;8(1):7848.

实验参考：

Cell Assay	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].
Animal Administration	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].
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]. Huang LE, et al. N-methyl D-aspartate receptor subtype 2B antagonist, Ro 25-6981, attenuates neuropathic pain by inhibiting postsynaptic density 95 expression. Sci Rep. 2018 May 18;8(1):7848.