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Lieferung & Zahlungsart

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Zuschläge

- Mindermengenzuschlag
- Trockeneiszuschlag
- Gefahrgutzuschlag
- Expressversand

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Tat-NR2B9c TFA

Cat. No.:	HY-P0117A
CAS No.:	1834571-04-8
Molecular Formula:	$C_{105}H_{188}N_{42}O_{30}.C_2HF_3O_2$
Molecular Weight:	2632.9
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 (TFA salt)

SOLVENT & SOLUBILITY

In Vitro

H_2O : ≥ 50 mg/mL (18.99 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	0.3798 mL	1.8990 mL	3.7981 mL
5 mM	0.0760 mL	0.3798 mL	0.7596 mL
10 mM	0.0380 mL	0.1899 mL	0.3798 mL

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

In Vivo

1. Add each solvent one by one: PBS
   Solubility: 100 mg/mL (37.98 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Tat-NR2B9c TFA (Tat-NR2Bct TFA) 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 TFA 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 TFA also inhibits neuronal nitric oxide synthase (nNOS)/PSD-95 interaction, and possesses neuroprotective efficacy[1].

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 nmol/g[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.
- Sci Rep. 2018 May 18;8(1):7848.
- Behav Brain Res. 7 January 2022, 113537.
- J Neuropathol Exp Neurol. 2020 Jul 1;79(7):800-808.
- PLoS One. 2020 Mar 3;15(3):e0229499.

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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.

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