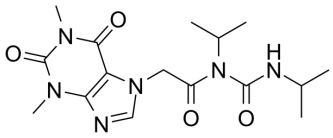
THX-B

Cat. No.:	HY-137322
CAS No.:	1372206-64-8
Molecular Formula:	$C_{16}H_{24}N_6O_4$
Molecular Weight:	364.4
Target:	Neurotensin Receptor
Pathway:	GPCR/G Protein; Neuronal Signaling
Storage:	4°C, protect from light * In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 100 mg/mL (274.42 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.7442 mL	13.7212 mL	27.4424 mL
	5 mM	0.5488 mL	2.7442 mL	5.4885 mL
	10 mM	0.2744 mL	1.3721 mL	2.7442 mL

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

BIOLOGICAL ACTIVITY

Description

THX-B is a potent and non-peptidic $p75^{NTR}$ (neurotrophin receptor p75) antagonist. THX-B can be used in the research of diabetic kidney disease, neurodegenerative and inflammatory disorders[1][2][3].

In Vitro

THX-B (10 μM, 4 days) decreases proliferation of myoblasts[1].
THX-B (10 μM, 1 h) inhibits NGF-induced phosphorylation of ERK1/2 in C2C12 myoblasts[1].
THX-B (20 μM, 24 h) decreases photoreceptor cell death and reactive gliosis in cultured rd10 retinas[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	C2C12 myoblasts
Concentration:	10 μM
Incubation Time:	Pre-treated for 1 hour
Result:	Inhibited βNGF-induced ERK2 phosphorylation by 67%. Inhibited proNGF-induced ERK2 phosphorylation by 90%.

Immunofluorescence[1]

Cell Line:	Cultured P22 rd10 retinas.
Concentration:	20 μM
Incubation Time:	24 h
Result:	Attenuated the thickening and enlargement of processes of astrocytes and Müller glia cells.

In Vivo

THX-B (50 μg in 125 μL PBS, i.p. weekly for 4 weeks) improves bladder function in a mouse model of diabetic voiding dysfunction[3].
THX-B (2 μL of 2 μg/μL, IVT injection, a single dose) elicits a neuroprotective effect on photoreceptor cells in P17 rd10 mice[2].
THX-B (40 μg in 20 μL, IVT injection) resolves the inflammatory, vascular, and neurodegenerative phases of the retinal pathology[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Mouse model of diabetic voiding dysfunction
Dosage:	50 μg in 125 μL PBS
Administration:	Intraperitoneal injection (i.p.)
Result:	Prevented bladder weight increase, which was 18% (95% CI 3%, 32%) and 37% (95% CI 14%, 60%) lower after 2 and 4 weeks of treatment.

Animal Model:	P17 rd10 mice[1]
Dosage:	2 μL of 2 μg/μL, single dose
Administration:	Intravitreal (IVT) injected in one eye
Result:	Increased the number of photoreceptor rows as well as the ONL/INL ratio. Decreased the total number of microglial cells in the treated retinas, as well as some of the inflammatory signs, such as GFAP, α2M and the proinflammatory cytokines IL-1β and TNF α.

REFERENCES

[1]. Abubakr H Mossa, et al. Antagonism of proNGF or its receptor p75 NTR reverses remodelling and improves bladder function in a mouse model of diabetic voiding dysfunction. Diabetologia. 2020 Sep;63(9):1932-1946.

[2]. Alba Galan, et al. Subconjunctival Delivery of p75NTR Antagonists Reduces the Inflammatory, Vascular, and Neurodegenerative Pathologies of Diabetic Retinopathy. Invest Ophthalmol Vis Sci. 2017 Jun 1;58(7):2852-2862.

[3]. María Platón-Corchado, et al. $p75^{NTR}$ antagonists attenuate photoreceptor cell loss in murine models of retinitis pigmentosa. Cell Death Dis. 2017 Jul 13;8(7):e2922.

[4]. Keren Ettinger, et al. Nerve growth factor stimulation of ERK1/2 phosphorylation requires both $p75^{NTR}$ and α9β1 integrin and confers myoprotection towards ischemia in C2C12 skeletal muscle cell model. Cell Signal. 2012 Dec;24(12):2378-88.

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

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