BT44

Cat. No.: HY-153175

CAS No.: 924759-42-2

Molecular Formula: $C_{28}H_{27}F_4N_3O_4S$

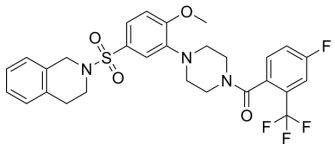
Molecular Weight: 577.59

Target: RET

Pathway: Protein Tyrosine Kinase/RTK

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	6 months
	-20°C	1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 62.5 mg/mL (108.21 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	1.7313 mL	8.6567 mL	17.3133 mL
	5 mM	0.3463 mL	1.7313 mL	3.4627 mL
	10 mM	0.1731 mL	0.8657 mL	1.7313 mL

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

BIOLOGICAL ACTIVITY

Description

BT44 is a selective RET activator. BT44 can penetrate through the blood-brain barrier and can be used for the research of neurodegenerative disorders and diabetes mellitus[1][2].

In Vitro

BT44 (7.5-75 μM; 15 min) promotes RET phosphorylation and selectively activates downstream cascades in the cells expressing GFL receptors[1].

BT44 (0.5-10 μM; 16-20 h) promotes neurite outgrowth from sensory neurons[1].

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

Western Blot Analysis[1]

Cell Line:	GFRα3-transfected MG87RET cells
Concentration:	7.5, 18, 35 and 75 μM
Incubation Time:	15 min
Result:	Increased RET and ERK phosphorylation.

In Vivo

BT44 (5-25 mg/kg; s.c.; every second day for 10, 42 or 14 days) alleviates sensory signs in the SNL and STZ models of neuropathic[1].

BT44 (12.5 or 25 mg/kg; s.c.; every second day for 10 days) protects IB4-positive neurons in DRGs of animals with experimental neuropathy[1].

BT44 (0.1 and 0.3 μg/24 h; infuse into the right dorsal striatum for 14 days) reverses amphetamine-induced motor imbalance and seems to protect dopaminergic fibers in the striatum in 6-OHDA rat model of Parkinson's disease[2].

BT44 (10 mg/kg; i.v.) penetrates the blood-brain barrier and is rapidly eliminated from the circulation (half-life ($t_{1/2}$) = 0.72 h) and brain ($t_{1/2}$ = 0.47 h) in rats[2].

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

Animal Model:	Wistar rats, spinal nerve ligation (SNL) and Streptozotocin (STZ; HY-13753)-induced diabetes mellitus models[1]
Dosage:	5, 12.5 or 25 mg/kg
Administration:	Subcutaneous injecton, every second day for 10, 42 or 14 days
Result:	Alleviated mechanical allodynia in the SNL animals. Treatment with the dose of 5 mg/kg alleviated mechanical hyperalgesia in the STZ-treated animals, while the 12.5 mg/kg dose was not effective. Concentration of 5 mg/kg attenuated cold allodynia in the STZ-treated animals during the first two weeks while the effect of 12.5 mg/kg was not significant.

Animal Model:	Wistar rats, SNL-induced diabetes mellitus model[1]
Dosage:	12.5 or 25 mg/kg
Administration:	Subcutaneous injecton, every second day for 10 days
Result:	Led to a significant increase in the number of IB4 expressing neurons in the ipsilateral DRGs. The 12.5 mg/kg dose protected IB4-positive neurons from SNL-induced lesion.

REFERENCES

[1]. Viisanen H, et al. Novel RET agonist for the treatment of experimental neuropathies. Mol Pain. 2020 Jan-Dec;16:1744806920950866.

[2]. Renko JM, et al. Neuroprotective Potential of a Small Molecule RET Agonist in Cultured Dopamine Neurons and Hemiparkinsonian Rats. J Parkinsons Dis. 2021;11(3):1023-1046.

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