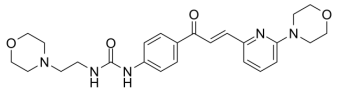
TRC051384

Cat. No.:	HY-101712
CAS No.:	867164-40-7
Molecular Formula:	$C_{25}H_{31}N_5O_4$
Molecular Weight:	465.54
Target:	HSP
Pathway:	Cell Cycle/DNA Damage; Metabolic Enzyme/Protease
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (214.80 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1480 mL	10.7402 mL	21.4804 mL
5 mM	0.4296 mL	2.1480 mL	4.2961 mL
10 mM	0.2148 mL	1.0740 mL	2.1480 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.37 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.37 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

TRC051384 is a potent inducer of heat shock protein 70 (HSP70). TRC051384 exhibits protective effects against neuronal trauma via inhibition of necroptosis. TRC051384 can be used for the research of ischemic stroke[1][2].

IC_{50} & Target

HSP70

In Vitro

TRC051384, dose dependently induces HSP70B mRNA by several hundred folds in both HeLa and rat primary mixed neurons. Treatment with TRC051384 results in significant dose-dependent increase in HSF1 transcriptional activity and recovery of luciferase activity. TRC051384 results in 60% inhibition at 6.25 μM and 90% inhibition at 12.5 μM of LPS-induced TNF-α expression in differentiated THP-1 cell line[1].

	MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	Treatment with TRC051384 significantly reduces stroke associated neuronal injury (87% reduction in area of penumbra recruited to infarct, and 25% reduction in brain edema) and disability in a rat model of transient ischemic stroke even when administered 8 hours post onset of ischemia. Significant improvement in survival (50% by day2 and 67.3% by day 7) is observed with TRC051384 treatment initiated at 4 hours after ischemia onset. Induction of HSP70 by TRC051384 involves HSF1 activation and results in elevated chaperone and anti-inflammatory activity[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [1]	HeLa cell transiently co-transfected with heat shock elements-luciferase reporter and normalization vector, β-galactosidase are treated with vehicle or TRC051384 (12.5 and 25 μM) for 4 hours. Cell lysates are then prepared and analyzed for luciferase and β-galactosidase activity[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Rats: Injured hemispheres from vehicle treated animals and TRC051384 treated animals are collected at 10-hour post-initiation of tMCAo. Total RNA from each brain sample is extracted followed by cDNA preparation. Each sample of cDNA is analyzed[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Clin Transl Med. 2023 Mar;13(3):e1229.
- Phytomedicine. 2023 Oct, 119, 154977.
- Domest Anim Endocrin. 2021 Jan;74:106533.

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REFERENCES

[1]. Chen T, et, al. HSP70 attenuates neuronal necroptosis through the HSP90α-RIPK3 pathway following neuronal trauma. Mol Biol Rep. 2023 Sep;50(9):7237-7244.

[2]. Mohanan A, et al. Delayed intervention in experimental stroke with TRC051384--a small molecule HSP70 inducer. Neuropharmacology. 2011 May;60(6):991-9.

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