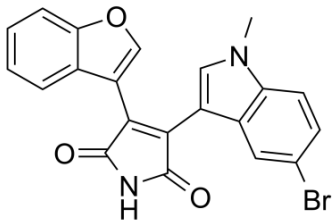
BIP-135

Cat. No.:	HY-111055
CAS No.:	941575-71-9
Molecular Formula:	$C_{21}H_{13}BrN_2O_3$
Molecular Weight:	421.24
Target:	GSK-3
Pathway:	PI3K/Akt/mTOR; Stem Cell/Wnt
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



BIOLOGICAL ACTIVITY

Description

BIP-135 is a potent and selective ATP-competitive GSK-3 inhibitor, with IC_{50}s of 16 nM and 21 nM for GSK-3α and GSK-3β, respectively. BIP 135 exhibits neuroprotective effect[1].

IC_{50} & Target

GSK-3α	GSK-3β
16 nM (IC_{50})	21 nM (IC_{50})

In Vitro

BIP-135 (20-30 μM; 72 hours) increases the survival motor neuron (SMN) protein levels at a dose of 25 μM in human SMA fibroblasts. And the typical bell-shaped dose-response curve is observed due to some toxicity at higher concentrations[1].
BIP-135 (20 μM; 48 hours) is a superior neuroprotective agent in the model of oxidative stress[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[1]

Cell Line:	Human SMA fibroblasts
Concentration:	20 μM, 25 μM, 30 μM
Incubation Time:	72 hours
Result:	Led to a 7-fold increase in SMN levels at 25 μM.

In Vivo

BIP-135 does not appear to be toxic and was well-tolerated by the animals (no decrease in body weight)[1].
BIP-135 (75 mg/kg; i.p.; daily; from postnatal day 0 to 21) prolongs the median survival time of Δ7 SMA KO mouse model of spinal muscular atrophy[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Male and female $SMN2^{+/+}$, $SMN2\Delta7^{+/+}$, $Smn^{+/-}$ mice[1]
Dosage:	75 mg/kg
Administration:	Intraperitoneal injection; daily; from postnatal day 0 to 21
Result:	Caused a modest extension in the median survival of SMA KO animals by two days.

REFERENCES

[1]. Chen PC, et al. Identification of a Maleimide-Based Glycogen Synthase Kinase-3 (GSK-3) Inhibitor, BIP-135, that Prolongs the Median Survival Time of Δ7 SMA KO Mouse Model of Spinal Muscular Atrophy. ACS Chem Neurosci. 2012 Jan 18;3(1):5-11.

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