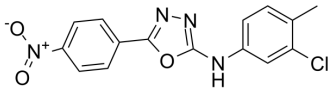
TC-G 24

Cat. No.:	HY-107529
CAS No.:	1257256-44-2
Molecular Formula:	$C_{15}H_{11}ClN_4O_3$
Molecular Weight:	330.73
Target:	GSK-3
Pathway:	PI3K/Akt/mTOR; Stem Cell/Wnt
Storage:	Please store the product under the recommended conditions in the Certificate of Analysis.



BIOLOGICAL ACTIVITY

Description

TC-G 24 (Compound 24) is a potent, selective glycogen synthase kinase-3β (GSK-3β) inhibitor with an IC_{50} of 17.1 nM. TC-G 24 can cross the BBB and can be used for studying many diseases such as type 2 diabetes mellitus, stroke, Alzheimer, and other related diseases[1].

IC_{50} & Target

GSK-3β
17.1 nM (IC_{50})

In Vitro

TC-G 24 (Compound 24) binds to the ATP binding site of GSK-3β[1].
TC-G 24 (1 μM, 4 h) blocks the FBW7α-mediated degradation of TPP1 in human embryonic kidney (HEK) 293T cells[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	293T cells
Concentration:	1 μM
Incubation Time:	4 h
Result:	Blocked the FBW7α-mediated degradation of TPP1

In Vivo

TC-G 24 (Compound 24) (0-15 mg/kg; i.p.; once) significantly raises liver glycogen content in a dose-dependent manner without obvious toxicity and can cross the BBB[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Six-week-old male C57BL/6N mice with weights averaging 22 g[1]
Dosage:	1, 5, and 15 mg/kg
Administration:	Intraperitoneal injection, once
Result:	Significantly raised liver glycogen content in a dose-dependent manner without obvious toxicity. Was detected in the brain at concentrations higher than in plasma for all three tested doses (38 ± 6, 113 ± 54 and 286 ± 58 ng/g brain tissue at 1, 5 and 15 mg/kg,

	respectively).

REFERENCES

[1]. Khanfar MA, et al. Discovery of novel GSK-3β inhibitors with potent in vitro and in vivo activities and excellent brain permeability using combined ligand- and structure-based virtual screening. J Med Chem. 2010 Dec 23;53(24):8534-45.

[2]. Lihui Wang, et al. FBW7 Mediates Senescence and Pulmonary Fibrosis through Telomere Uncapping. Cell Metab. 2020 Nov 3;32(5):860-877.e9.

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