Cu(II)GTSM

Cat. No.: HY-139324

CAS No.: 68341-14-0

Molecular Formula: $C_6H_{10}CuN_6S_2$

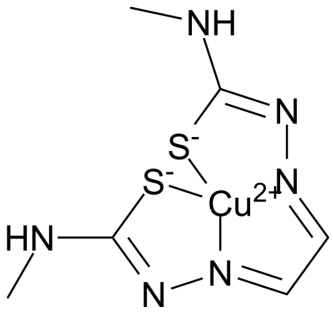
Molecular Weight: 293.86

Target: GSK-3; Amyloid-β

Pathway: PI3K/Akt/mTOR; Stem Cell/Wnt; Neuronal Signaling

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 : 6.67 mg/mL (22.70 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	3.4030 mL	17.0149 mL	34.0298 mL
	5 mM	0.6806 mL	3.4030 mL	6.8060 mL
	10 mM	0.3403 mL	1.7015 mL	3.4030 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: 0.67 mg/mL (2.28 mM); Clear solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: 0.67 mg/mL (2.28 mM); Clear solution; Need ultrasonic

BIOLOGICAL ACTIVITY

Description

Cu(II)GTSM, a cell-permeable Cu-complex, significantly inhibits GSK3β. Cu(II)GTSM inhibits Amyloid-β oligomers (AβOs) and decreases tau phosphorylation. Cu(II)GTSM also decreases the abundance of Amyloid-β trimers. Cu(II)GTSM is a potential anticancer and antimicrobial agent[1][2].

IC_{50} & Target		
	GSK3β	Amyloid-β oligomers

In Vitro

Cu(II)GTSM induces GSK3β phosphorylation at serine-9 (ser9) via its upstream kinase, protein kinase B (Akt), and aslo increases the phosphorylation of the associated extracellular signal-related kinase 1/2 (ERK1/2) in SH-SY5Y cells.
Tau phosphorylation at ser404 was decreased in CuII(gtsm)-treated cells by 64%[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Western Blot Analysis[2]

Cell Line:	SH-SY5Y cells
Concentration:	25 μM
Incubation Time:	2 hours
Result:	Inhibits GSK3β and decreases Tau phosphorylation.

In Vivo

Cu(II)GTSM decreases brain Aβ trimer levels in AD mice, and can reverse cognitive deficits in APP/PS1 transgenic AD mice[2].

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

Animal Model:	AD mice (K670N, M671L; 5-6 months old)[2]
Dosage:	10 mg/kg
Administration:	Daily; p.o.
Result:	Restores cognitive performance of the AD mice to levels expected for healthy, cognitively normal mice.

REFERENCES

[1]. Andres SA, et al. Synthesis, Characterization, and Biological Activity of Hybrid Thiosemicarbazone-Alkylthiocarbamate Metal Complexes. Inorg Chem. 2020;59(7):4924-4935.

[2]. Crouch PJ, et al. Increasing Cu bioavailability inhibits Abeta oligomers and tau phosphorylation. Proc Natl Acad Sci U S A. 2009;106(2):381-386.

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