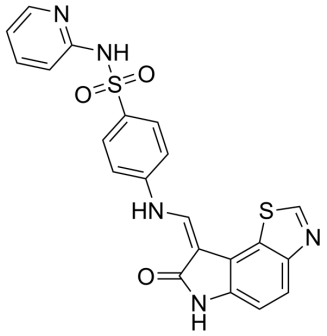
GW8510

Cat. No.:	HY-112358
CAS No.:	222036-17-1
Molecular Formula:	$C_{21}H_{15}N_5O_3S_2$
Molecular Weight:	449.51
Target:	CDK
Pathway:	Cell Cycle/DNA Damage
Storage:	Powder -20°C 3 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (55.62 mM; Need ultrasonic)

Preparing Stock Solutions	Mass / Solvent / Concentration	1 mg	5 mg	10 mg
	1 mM	2.2246 mL	11.1232 mL	22.2464 mL
	5 mM	0.4449 mL	2.2246 mL	4.4493 mL
	10 mM	0.2225 mL	1.1123 mL	2.2246 mL

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

BIOLOGICAL ACTIVITY

Description

GW8510 is a potent cyclin-dependent kinase-2 (CDK2) inhibitor. GW8510 is also a ribonucleotide reductase M2 (RRM2) inhibitor. GW8510 exhibits neuroprotective and anticancer activities[1][2][3].

IC_{50} & Target	CDK2	CDK5	RRM2

In Vitro

GW8510 (0.5-4 μM; 72 h) inhibits viability of HCT116 cells in a dose-dependent manner[2].

GW8510 (1-4 μM; 24 h) inhibits RRM2 expression without alteration of RRM1 expression[2].

GW8510 inhibits CDK2 and other CDKs when tested in in vitro biochemical assays, when used on cultured neurons it only inhibits CDK5[1].

GW8510 inhibits the death of cerebellar granule neurons caused by switching them from high potassium medium to low potassium medium[1].

Combination with GW8510 (5 μM; 48 h) and Tamoxifen (5 μM; 48 h) significantly inhibits survival of the Tamoxifen-resistant breast cancer cells (BBCs) through induction of autophagic cell death[3].

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

Cell Viability Assay[2]

Cell Line:	HCT116 cells
Concentration:	0.5, 1, 2, 4 μM
Incubation Time:	72 hours
Result:	Inhibited HCT116 cells growth.

Western Blot Analysis[2]

Cell Line:	HCT116 cells
Concentration:	1, 2, 4 μM
Incubation Time:	24 hours
Result:	Inhibited RRM2 expression. The reduction of RRM2 protein level can be reversed by MG132.

In Vivo

Combination with GW8510 and Tamoxifen enhances tumoricidal effect on Tamoxifen-resistant BBC xenograft through autophagy induction[3].

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

REFERENCES

[1]. ARCAMONE F, et, al. STRUCTURE AND SYNTHESIS OF DISTAMYCIN A. Nature. 1964 Sep 5;203:1064-5.

[2]. Hiraku Y, et, al. Distamycin A, a minor groove binder, changes enediyne-induced DNA cleavage sites and enhances apoptosis. Nucleic Acids Res Suppl. 2002;(2):95-6.

[3]. Majumder P, et, al. Effect of DNA groove binder distamycin A upon chromatin structure. PLoS One. 2011;6(10):e26486.

Tel: 609-228-6898 Fax: 609-228-5909 E-mail: tech@MedChemExpress.com

Address: 1 Deer Park Dr, Suite Q, Monmouth Junction, NJ 08852, USA