E7016

Cat. No.: HY-13540

CAS No.: 902128-92-1

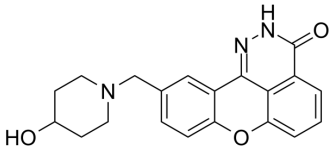
Molecular Formula: $C_{20}H_{19}N_3O_3$

Molecular Weight: 349.38

Target: PARP

Pathway: Cell Cycle/DNA Damage; Epigenetics

Storage: 4°C, protect from light
* In solvent : -80°C, 6 months; -20°C, 1 month (protect from light)



SOLVENT & SOLUBILITY

In Vitro

DMSO : 25 mg/mL (71.56 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.8622 mL	14.3111 mL	28.6221 mL
	5 mM	0.5724 mL	2.8622 mL	5.7244 mL
	10 mM	0.2862 mL	1.4311 mL	2.8622 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 1.25 mg/mL (3.58 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 1.25 mg/mL (3.58 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

E7016 (GPI 21016) is an orally available PARP inhibitor. E7016 can enhance tumor cell radiosensitivity in vitro and in vivo through the inhibition of DNA repair. E7016 acts as a potential anticancer agent[1][2].

IC_{50} & Target

PARP

In Vitro

E7016 can enhance tumor cell radiosensitivity through the inhibition of DNA repair[1].
E7016 (3 μM)-mediated radiosensitization occurs through an increase in the number of cells undergoing mitotic catastrophe and not an increase in the number of cells undergoing apoptosis[1].
E7016 inhibits PARP by mimicking NAD^+[2].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Apoptosis Analysis[1]

Cell Line:	The U251 human glioblastoma cell line
Concentration:	3 μM
Incubation Time:	6 hours prior to irradiation and were stained at 24 and 72 h postirradiation
Result:	The number of cells in mitotic catastrophe was significantly greater in the E7016-treated irradiated cells than in cells that received radiation only at 24 hours postirradiation.

In Vivo

E7016 has antitumor efficacy in murine xenograft studies[1].
Administration of E7016 (40 mg/kg; oral gavage) to mice bearing U251 xenografts enhances the effectiveness of the Temozolomide/radiation combination[1].
Mice treated with E7016/irradiation/Temozolomide have an additional growth delay of six days compared with the combination of Temozolomide and irradiation in vivo[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	Four- to six-week-old female nude mice[3]
Dosage:	40 mg/kg
Administration:	Oral gavage
Result:	E7016 enhanced the radiation/Temozolomide (3 mg/kg orally)-induced tumor growth delay of U251 xenografts.

REFERENCES

[1]. Andrea L Russo, et al. In vitro and in vivo radiosensitization of glioblastoma cells by the poly (ADP-ribose) polymerase inhibitor E7016. Clin Cancer Res. 2009 Jan 15;15(2):607-12.

[2]. W George Lai, et al. A Baeyer-Villiger oxidation specifically catalyzed by human flavin-containing monooxygenase 5. Drug Metab Dispos. 2011 Jan;39(1):61-70.

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