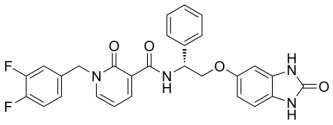
MP7

Cat. No.:	HY-14440
CAS No.:	1001409-50-2
Molecular Formula:	$C_{28}H_{22}F_2N_4O_4$
Molecular Weight:	516.5
Target:	PDK-1
Pathway:	PI3K/Akt/mTOR
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 2 years -20°C 1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (193.61 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9361 mL	9.6805 mL	19.3611 mL
5 mM	0.3872 mL	1.9361 mL	3.8722 mL
10 mM	0.1936 mL	0.9681 mL	1.9361 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: ≥ 2.75 mg/mL (5.32 mM); Clear solution
2. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.75 mg/mL (5.32 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

MP7 (PDK1 inhibitor) is a phosphoinositide-dependent kinase-1 (PDK1) inhibitor.

IC_{50} & Target

PDK1[1]

In Vitro

Cell counting of U87MG-derived glioma stem cells (GSCs) confirms that Alisertib and, to a minor extent, MP7 (PDK1 inhibitor) are able to decrease the number of viable cells. When combined together, GSC viability is further reduced with respect to single-treated cells. As observed in U87MG cells, when used at the highest concentrations (i.e., 1.5 μM Alisertib and 2.5 μM MP7), a significant enhancement in the number of dead cells is evidenced. Following 72 h treatment, MP7 alone does not show a significant inhibition of glioblastoma multiforme (GBM) proliferation. MP7 has been shown to have only minimal

effects on monolayer cell growth in several cancer cell lines, with IC_{50} values in the micromolar range[1].

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

PROTOCOL

Cell Assay [1]

The human GBM cells (i.e., U87MG, U343MG, or ANGM-CSS) or the respective GSCs are seeded and incubated for the indicated times with the indicated concentrations of SA16 (1 nM to 100 μM), MP7 (2.5 nM, 25 nM, 250 nM and 2.5 μM), or Alisertib. When indicated, cells are treated with MP7 and Alisertib in combination. To verify GSC chemoresistance, U87MG or GSCs are incubated with 50 μM TMZ for 72 h. For the long-term treatment of cells, NSC or complete medium containing drugs is replaced every 3 days. Cell proliferation is determined using the MTS assay: the dehydrogenase activity in active mitochondria reduces MTS to the soluble formazan product, whose absorbance at 490 nm is measured with an automated plate reader. The mean background from each test condition is subtracted, and the data are expressed as the percentage of untreated cells (control). IC_{50} values are derived from the sigmoid dose-response curve. The percentage of inhibition is calculated as 100% minus the percentage of cell proliferation[1].

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

REFERENCES

[1]. Daniele S, et al. Dual Inhibition of PDK1 and Aurora Kinase A: An Effective Strategy to Induce Differentiation and Apoptosis of Human Glioblastoma Multiforme Stem Cells. ACS Chem Neurosci. 2017 Jan 18;8(1):100-114.

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