Ipatasertib

Cat. No.: HY-15186

CAS No.: 1001264-89-6

Molecular Formula: $C_{24}H_{32}ClN_5O_2$

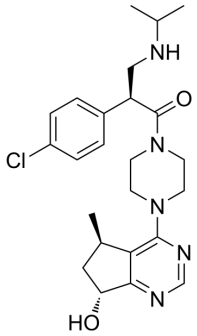
Molecular Weight: 458.00

Target: Akt; Apoptosis; Organoid

Pathway: PI3K/Akt/mTOR; Apoptosis; Stem Cell/Wnt

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 (218.34 mM; Need ultrasonic)

H_2O : 3.57 mg/mL (7.79 mM; ultrasonic and warming and heat to 60°C)

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.1834 mL	10.9170 mL	21.8341 mL
5 mM	0.4367 mL	2.1834 mL	4.3668 mL
10 mM	0.2183 mL	1.0917 mL	2.1834 mL

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

In Vivo

1. Add each solvent one by one: 0.5% Methyl cellulose/0.5% Tween-80 in Saline water
   Solubility: 10 mg/mL (21.83 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 5 mg/mL (10.92 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 5 mg/mL (10.92 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.08 mg/mL (4.54 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Ipatasertib (GDC-0068) is an orally active, highly selective and ATP-competitive pan-Akt inhibitor with IC_{50} values of 5, 18, 8 nM for Akt1/2/3, respectively. Ipatasertib synchronously activates FoxO3a and NF-κB through inhibition of Akt leading to p53-independent activation of PUMA. Ipatasertib also induces apoptosis in cancer cells and inhibits tumor growth in xenograft mouse models[1][2].

IC_{50} & Target	Akt1	Akt3	Akt2	PKA
	5 nM (IC_{50})	8 nM (IC_{50})	18 nM (IC_{50})	3100 nM (IC_{50})

In Vitro

Ipatasertib (10 μM; 12, 24 h) suppresses colon cancer cell proliferation by p53 irrespectively activating PUMA in vitro[1].
Ipatasertib (1, 5, 10, 20 μM; 24 h/10 μM; 3, 6, 12, 24 h) up-regulates the expression level of PUMA in a concentration and time dependent manner in HCT116 cells[1].
Ipatasertib increases the mRNA level of PUMA in HCT116 WT, $p53^{-/-}$, and DLD1 (p53 mutant) cells[1].
Ipatasertib (10 μM; 24 h) induces apoptosis through PUMA/Bax pathway in HCT116 cells[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Viability Assay[1]

Cell Line:	HCT116 WT, $p53^{-/-}$, and DLD1 (p53 mutant) cells
Concentration:	10 μM
Incubation Time:	12, 24 h
Result:	Decreased all the three cell lines viability.

Apoptosis Analysis[1]

Cell Line:	HCT116 cells
Concentration:	10 μM
Incubation Time:	24 h
Result:	Induced apoptosis through PUMA/Bax pathway.

Western Blot Analysis[1]

Cell Line:	HCT116 cells
Concentration:	1, 5, 10, 20 μM for 24 h/10 μM for 3, 6, 12, 24 h
Incubation Time:	24 h; 3, 6, 12, 24 h
Result:	Increased the level of PUMA in a concentration and time dependent manner.

In Vivo

Ipatasertib (30 mg/kg; p.o.; single daily for 15 consecutive days) exhibits PUMA-dependent antitumor activity in HCT116 WT and $PUMA^{-/-}$ cells xenograft nude mice model[1].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Animal Model:	HCT116 WT and $PUMA^{-/-}$ cells xenograft nude mice model[1].
Dosage:	30 mg/kg
Administration:	Oral gavage; single daily for 15 consecutive days.
Result:	Inhibited growth of tumors in a PUMA-dependent manner.

CUSTOMER VALIDATION

- Cell Metab. 2021 Nov 2;33(11):2247-2259.e6.

- Nat Cell Biol. 2025 Jan 8.
- Blood. 2023 May 26;blood.2022018752.
- Sci Transl Med. 2018 Jul 18;10(450):eaaq1093.
- Nat Commun. 2025 Feb 25;16(1):1774.

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REFERENCES

[1]. Sun L, et al. Ipatasertib, a novel Akt inhibitor, induces transcription factor FoxO3a and NF-κB directly regulates PUMA-dependent apoptosis. Cell Death Dis. 2018 Sep 5;9(9):911.

[2]. Blake JF, et al. Discovery and preclinical pharmacology of a selective ATP-competitive Akt inhibitor (GDC-0068) for the treatment of human tumors. J Med Chem. 2012 Sep 27;55(18):8110-27.

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