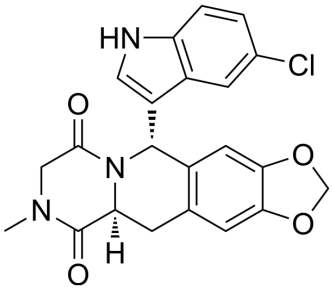
ISA-2011B

Cat. No.:	HY-16937
CAS No.:	1395347-24-6
Molecular Formula:	$C_{22}H_{18}ClN_3O_4$
Molecular Weight:	423.85
Target:	Others
Pathway:	Others
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 (235.93 mM; Need ultrasonic)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	1 mM	2.3593 mL	11.7966 mL	23.5933 mL
	5 mM	0.4719 mL	2.3593 mL	4.7187 mL
	10 mM	0.2359 mL	1.1797 mL	2.3593 mL

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

In Vivo

1. Add each solvent one by one: 0.5% CMC-Na/saline water
   Solubility: 4 mg/mL (9.44 mM); Suspended solution; Need ultrasonic
2. Add each solvent one by one: 10% DMSO >> 40% PEG300 >> 5% Tween-80 >> 45% saline
   Solubility: ≥ 2.5 mg/mL (5.90 mM); Clear solution
3. Add each solvent one by one: 10% DMSO >> 90% (20% SBE-β-CD in saline)
   Solubility: ≥ 2.5 mg/mL (5.90 mM); Clear solution
4. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (5.90 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

ISA-2011B is a PIP5K1α inhibitor with promising anticancer effects .

In Vitro

The proliferation rate of PC-3 cells after treatment with ISA-2011B at 10, 20, and 50 μM is significantly reduced to 58.77%, 48.65%, and 21.62% of vehicle-treated controls, respectively. ISA-2011B exhibits the highest binding affinity to PIP5K1α, and to MAP/microtubule affinity-regulating kinase 1 and 4 (MARK1 and MARK4) across 460 kinases. ISA-2011B treatment inhibits

	PIP5K1α expression by 78.6% in PC-3 cells[1]. ISA-2011B leads to a remarkable reduction in AR-V7 and CDK1 in both nucleus and cytoplasm of 22Rv1 cells. ISA-2011B treatment also abolishes AR expression in the nucleus, without depleting the cytoplasmic AR[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
In Vivo	ISA-2011B significantly inhibits growth of tumor cells in xenograft mice, and is mediated by targeting PIP5K1α-associated PI3K/AKT and the downstream survival, proliferation, and invasion pathways[1]. Overexpression of AR-V7 increases PIP5K1α, promotes rapid growth of PCa in xenograft mice, whereas inhibition of PIP5K1α by its inhibitor ISA-2011B suppresses the growth and invasiveness of xenograft tumors overexpressing AR-V7. ISA-2011B disrupts protein stabilization of AR-V7 which is dependent on PIP5K1α, leading to suppression of invasive growth of AR-V7-high tumors in xenograft mice[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

PROTOCOL

Cell Assay [2]	Cells are grown in phenol red-free RPMI-1640 medium 24 hours and then are treated with drugs alone or in combination for 24 hours or 48 hours. MDV3100 at 5 μM or ISA-2011B at 20 μM or 50 μM final concentrations or solvent DMSO 1% is used. For treatment of 22Rv1 cells with MG132, a proteasome inhibitor, cells are treated with MG132 at 1 μM. For combination treatment of MG132 and ISA-2011B, cells are pre-treated with MG132 for 30 min at 1 μM prior to treatment of ISA-2011B[2]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.
Animal Administration [1]	Mice: BALB/c nude mice aged 8 to 12 wk are used in the experiments. Tumor cells are implanted into the mice. Tumor xenografts are treated with vehicle (control), RP-56976 (10 mg/kg), ISA-2011B (40 mg/kg), and RP-56976 (10 mg/kg) in combination with ISA-2011B (40 mg/kg) every second day[1]. MCE has not independently confirmed the accuracy of these methods. They are for reference only.

CUSTOMER VALIDATION

- Sci Adv. 2022 Jul 22;8(29):eabn1440.
- Sci Adv. 2019 Mar 27;5(3):eaat4872.
- Cell Rep. 2023, 42(1): 111905.
- FEBS J. 2020 Jul;287(14):3042-3064.
- J Cell Mol Med. 2018 Sep;22(9):4117-4129.

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REFERENCES

[1]. Semenas J, et al. The role of PI3K/AKT-related PIP5K1α and the discovery of its selective inhibitor for treatment of advanced prostate cancer. Proc Natl Acad Sci U S A. 2014 Sep 2;111(35):E3689-98.

[2]. Sarwar M, et al. Targeted suppression of AR-V7 using PIP5K1α inhibitor overcomes MDV3100 resistance in prostate cancer cells. Oncotarget. 2016 Sep 27;7(39):63065-63081.

Caution: Product has not been fully validated for medical applications. For research use only.

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