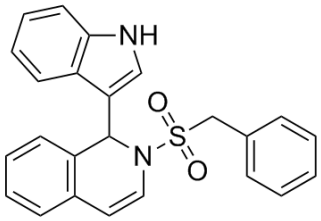
IBR2

Cat. No.:	HY-103710
CAS No.:	313526-24-8
Molecular Formula:	$C_{24}H_{20}N_2O_2S$
Molecular Weight:	400.49
Target:	RAD51; Apoptosis
Pathway:	Cell Cycle/DNA Damage; Apoptosis
Storage:	Powder -20°C 3 years 4°C 2 years In solvent -80°C 6 months -20°C 1 month



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 100 mg/mL (249.69 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4969 mL	12.4847 mL	24.9694 mL
5 mM	0.4994 mL	2.4969 mL	4.9939 mL
10 mM	0.2497 mL	1.2485 mL	2.4969 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: ≥ 2.5 mg/mL (6.24 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

IBR2 is a potent and specific RAD51 inhibitor and inhibits RAD51-mediated DNA double-strand break repair. IBR2 disrupts RAD51 multimerization, accelerates proteasome-mediated RAD51 protein degradation, inhibits cancer cell growth and induces apoptosis[1][2].

IC_{50} & Target

RAD51[1]

In Vitro

IBR2 shows interesting RAD51 inhibition activities. RAD51 is rapidly degraded in IBR2-treated cancer cells, and the homologous recombination repair is impaired, subsequently leading to cell death. The IC_{50} values of the original IBR2 are in the range of 12-20 μM for most tested cancer cell lines. IBR2 can inhibit the growth of triple-negative human breast cancer cell line MBA-MD-468 with an IC_{50} of 14.8 μM[1].

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

PROTOCOL

Cell Assay [1]

Human breast cancer cell lines MCF7, MDA-MB-231, MDA-MB-361, MDA-MB-435, MDA-MB468, Hs578-T, human osteosarcoma cell line U20S, human glioblastoma cell line T98G and human cervical adenocarcinoma cell line HeLa are used. Standard XTT assays with a four-day drug treatment procedure are performed to measure the dose dependent cytotoxicity of IBR analogs in cultured cells. In brief, cells are plated on 96-well dishes one day before the drug treatment, followed by drug (e.g., IBR2) treatment on day 2 and XTT assay on day 6 after drug addition by using a commercial cell proliferation kit . Triplicate sets are measured and compiled for final data presentation[1].

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CUSTOMER VALIDATION

- Biological Sciences. 2020 Sep.

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REFERENCES

[1]. Zhu J, et al. Synthesis, molecular modeling, and biological evaluation of novel RAD51 inhibitors. Eur J Med Chem. 2015;96:196-208.

[2]. Jiewen Zhu, et al. A novel small molecule RAD51 inactivator overcomes imatinib-resistance in chronic myeloid leukaemia. EMBO Mol Med. 2013 Mar;5(3):353-65.

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