BOLD-100

Cat. No.: HY-16350

CAS No.: 197723-00-5

Molecular Formula: $C_{14}H_{12}Cl_4N_4NaRu$

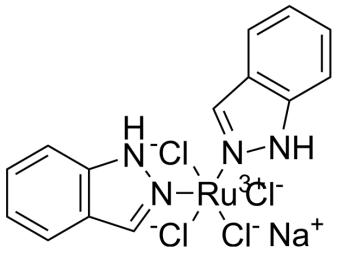
Molecular Weight: 502.14

Target: DNA/RNA Synthesis; Apoptosis

Pathway: Cell Cycle/DNA Damage; Apoptosis

Storage: 4°C, sealed storage, away from moisture

* In solvent : -80°C, 6 months; -20°C, 1 month (sealed storage, away from moisture)



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 59 mg/mL (117.50 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	1.9915 mL	9.9574 mL	19.9148 mL
5 mM	0.3983 mL	1.9915 mL	3.9830 mL
10 mM	0.1991 mL	0.9957 mL	1.9915 mL

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

BIOLOGICAL ACTIVITY

Description

BOLD-100 (NKP-1339; IT-139) is the first-in-class ruthenium-based anticancer agent in development against solid cancer with limited side effects. BOLD-100 induces G2/M cell cycle arrest, blockage of DNA synthesis, and induction of apoptosis via the mitochondrial pathway. BOLD-100 has a high tumor targeting potential, strongly binds to serum proteins such as albumin and transferrin and activates in the reductive tumor milieu[1].

In Vitro

BOLD-100 (0-200 μM; 72 hours) has the anticancer activity against malignant cell lines of diverse origin, exhibits IC_{50} values of 45-200?μM for KP1339 mono-therapy. It against Hepatoma cell line, Hep3B, HepG2, PLC/PRF/5 and HCC2 cells with the Mean IC_{50} value of 186.3 μM, 165.4 μM, 124.4 μM, and 69.4 μM, respectively. It against Melanoma cell line, VM-1, VM-21,VM-48 with IC_{50} values of 178 μM, 111 μM, and 143 μM, respectively. It against Lung cancer and Colon cancer cell lines, inhibits A549, VL-8, SW480 and HCT116 cells, respectively[2].

BOLD-100 (0-150 μM; 24 hours) induces cell apoptosis alone. When it combines with sorafenib, it increases the numbers of the apopotic cells. Additionally, the p-PARP and caspase 7 cleavage is promoted either[2].

BOLD-100 (0-150 μM; 24 hours) can promote phosphorylation of STAT3 and CREB expression, however, the decreasation is inhibited by sorafenib cotreatment[2].

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

Cell Viability Assay[2]

Cell Line:	Hepatoma, Melanoma, Lung cancer and Colon cancer cell lines
Concentration:	0 μM, 50 μM, 100 μM, 150 μM and 200 μM
Incubation Time:	72 hours
Result:	Has anti-cancer activity in diverse malignant tumour cell types.

Apoptosis Analysis[2]

Cell Line:	Hep3B cells
Concentration:	0 μM, 75 μM, 150 μM
Incubation Time:	72 hours
Result:	Promoted cell apoptosis as a concentration manner.

Western Blot Analysis[2]

Cell Line:	Hep3B cells
Concentration:	0 μM, 75 μM, 150 μM
Incubation Time:	72 hours
Result:	Increased p-STAT3 and p-CREB expression in cells without sorafenib cotreatment.

In Vivo

BOLD-100 (intravenous injection; 30 mg/kg; once a week; 42sdays) combines with the multi-kinase inhibitor sorafenib and exhibits a further anticancer activity when compares to the BOLD-100 treatment alone in Hep3B xenografts grown in Balb/c SCID mice [2].

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

Animal Model:	Hep3B xenograft in Balb/c mice[2]
Dosage:	30 mg/kg
Administration:	Intravenous injection
Result:	Had synergistic activity of KP1339 with sorafenib in vivo.

REFERENCES

[1]. Robert Trondl, et al. NKP-1339, the first ruthenium-based anticancer drug on the edge to clinical application. Chemical Science. Chemical Science

[2]. Heffeter P, et al. The ruthenium compound KP1339 potentiates the anticancer activity of sorafenib in vitro and in vivo. Eur J Cancer. 2013 Oct;49(15):3366-75.

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