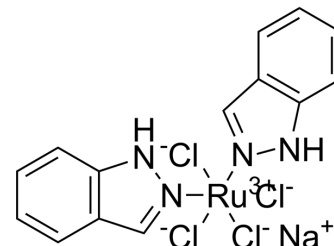


BOLD-100

Cat. No.:	HY-16350
CAS No.:	197723-00-5
Molecular Formula:	C ₁₄ H ₁₂ Cl ₄ N ₄ NaRu
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 Concentration	Mass		
		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 IC₅₀ 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 IC₅₀ 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 IC₅₀ 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 apoptotic 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 decrease 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 μ M, 50 μ M, 100 μ M, 150 μ M and 200 μ M
	Incubation Time:	72 hours
	Result:	Has anti-cancer activity in diverse malignant tumour cell types.
	Apoptosis Analysis ^[2]	
	Cell Line:	Hep3B cells
	Concentration:	0 μ M, 75 μ M, 150 μ M
	Incubation Time:	72 hours
	Result:	Promoted cell apoptosis as a concentration manner.
	Western Blot Analysis ^[2]	
	Cell Line:	Hep3B cells
	Concentration:	0 μ M, 75 μ M, 150 μ M
	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.

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

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