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Lieferung & Zahlungsart

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Zuschläge

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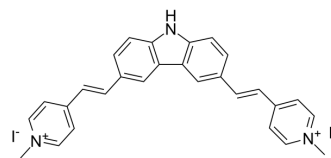
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BMVC

Cat. No.:	HY-135775
CAS No.:	627810-06-4
Molecular Formula:	C ₂₈ H ₂₅ I ₂ N ₃
Molecular Weight:	657.33
Target:	G-quadruplex; Telomerase; DNA/RNA Synthesis
Pathway:	Cell Cycle/DNA Damage
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 : 10 mg/mL (15.21 mM; ultrasonic and warming and heat to 60°C)

	Solvent Concentration	Mass	1 mg	5 mg	10 mg
Preparing Stock Solutions	1 mM		1.5213 mL	7.6065 mL	15.2131 mL
	5 mM		0.3043 mL	1.5213 mL	3.0426 mL
	10 mM		0.1521 mL	0.7607 mL	1.5213 mL

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

BIOLOGICAL ACTIVITY

Description

BMVC is a potent G-quadruplex (G4) stabilizer and a selective telomerase inhibitor with an IC₅₀ of ~0.2 μM. BMVC inhibits Taq DNA polymerase with an IC₅₀ of ~2.5 μM. BMVC increases the melting temperature of G4 structure of telomere and accelerates telomere length shortening. Anticancer activities^{[1][2]}.

IC₅₀ & Target

IC₅₀: ~0.2 μM (Telomerase)^[1]
G-quadruplex^[1]
IC₅₀: ~2.5 μM (Taq DNA polymerase)^[1]

In Vitro

BMVC (0.5 μM; 0-18 days; H1299 cells) treatment markedly increases the percentage of sub-G1-phase cells after 18 days^[1]. BMVC (0.5 μM; 0-18 days; H1299 cells) long-term treatment leads to ceasing of cell growth and eventually cell death through apoptosis. The long-term BMVC treatment induces senescence program in H1299 cells^[1]. In BMVC-treated cancer cells, hallmarks of senescence, including morphologic changes, detection of senescence-associated β-galactosidase activity, and decreased bromodeoxyuridine incorporation, are detected. The BMVC-induced senescence phenotype is accompanied by progressive telomere shortening and detection of the DNA damage foci, indicating that BMVC caused telomere uncapping after long-term treatments^[1].

BMVC also suppresses the tumor-related properties of cancer cells, including cell migration, colony-forming ability, and anchorage-independent growth^[1].

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

Cell Cycle Analysis^[1]

Cell Line:	H1299 cells
Concentration:	0.5 μ M
Incubation Time:	0 day, 6 days, 12 days, 18 days
Result:	The percentage of sub-G1-phase cells was markedly increased after 18 days.

Apoptosis Analysis^[1]

Cell Line:	H1299 cells
Concentration:	0.5 μ M
Incubation Time:	0 day, 6 days, 12 days, 18 days
Result:	Increased apoptotic cells.

In Vivo

BMVC (1 mg/kg; intraperitoneal injection; every 3 day; BALB/cAnN.Cg-Foxn1^{nu}/CrINarl mice) treatment delays tumorigenic potential of cancer cells in vivo^[1].

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

Animal Model:	BALB/cAnN.Cg-Foxn1 ^{nu} /CrINarl mice injected with H1299 cells ^[1]
Dosage:	1 mg/kg
Administration:	Intraperitoneal injection; every 3 day
Result:	The growth rates of tumors in animals were significantly slower than that of control animals. The tumor cells of the mice were indeed entering apoptosis.

CUSTOMER VALIDATION

- Nucleic Acids Res. 2021 Dec 16;49(22):12634-12643.

See more customer validations on www.MedChemExpress.com

REFERENCES

[1]. Huang FC, et al. G-quadruplex stabilizer 3,6-bis(1-methyl-4-vinylpyridinium)carbazole diiodide induces accelerated senescence and inhibits tumorigenic properties in cancer cells. Mol Cancer Res. 2008 Jun;6(6):955-64.

[2]. Jen-Fei Chu, et al. A Novel Method for Screening G-quadruplex Stabilizers to Human Telomeres. Journal of the Chinese Chemical Society, 2011, 58, 296-300.

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

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