BMVC

Cat. No.: HY-135775

CAS No.: 627810-06-4

Molecular Formula: $C_{28}H_{25}I_2N_3$

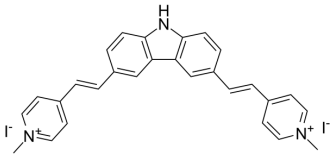
Molecular Weight: 657.33

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)

Preparing Stock Solutions	Solvent / Mass / Concentration	1 mg	5 mg	10 mg
	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 IC_{50} of ~0.2 μM. BMVC inhibits Taq DNA polymerase with an IC_{50} of ~2.5 μM. BMVC increases the melting temperature of G4 structure of telomere and accelerates telomere length shortening. Anticancer activities[1][2].

IC_{50} & 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 decreasesd 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 μM
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 μM
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}/CrlNarl 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}/CrlNarl 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.

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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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