Bromosporine

Cat. No.: HY-15815

CAS No.: 1619994-69-2

Molecular Formula: $C_{17}H_{20}N_6O_4S$

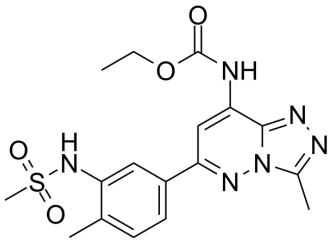
Molecular Weight: 404.44

Target: Epigenetic Reader Domain; Apoptosis; CDK; HIV

Pathway: Epigenetics; Apoptosis; Cell Cycle/DNA Damage; Anti-infection

Storage:

Powder	-20°C	3 years
	4°C	2 years
In solvent	-80°C	2 years
	-20°C	1 year



SOLVENT & SOLUBILITY

In Vitro

DMSO : ≥ 51.7 mg/mL (127.83 mM)

* "≥" means soluble, but saturation unknown.

Preparing Stock Solutions

Solvent / Mass / Concentration	1 mg	5 mg	10 mg
1 mM	2.4726 mL	12.3628 mL	24.7255 mL
5 mM	0.4945 mL	2.4726 mL	4.9451 mL
10 mM	0.2473 mL	1.2363 mL	2.4726 mL

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

In Vivo

1. Add each solvent one by one: 10% DMSO >> 90% corn oil
   Solubility: ≥ 2.5 mg/mL (6.18 mM); Clear solution

BIOLOGICAL ACTIVITY

Description

Bromosporine is a potent BET inhibitor with an IC_{50} value of 2.1 μM for PCAF. Bromosporine can arrest cell cycle and induce apoptosis in cancer cells. Bromosporine exhibits excellent antitumor activity in xenograft mice model when combined with 5-FU (HY-90006). Bromosporine can increase CDK9 T-loop phosphorylation in HIV-1 latency models, resulting the protection of reactivate HIV-1 replication from latency. Bromosporine can be used to research colorectal cancer, acute myeloid leukemia (AML) and AIDS[1][2][3][4].

IC_{50} & Target

IC_{50}: 2.1 μM (PCAF)[2]
Apoptosis, BET, CDK9, HIV-1[1][3][4]

In Vitro

Bromosporine (0-1000 nM; 72 h) synergistically inhibits cell growth in CRC cells with 5-FU (HY-90006)[1].
Bromosporine (various concentration; 48 h) causes a distinct increase in the cells arrested at G1 phase when combined with

5-FU[1].
Bromosporine (various concentration; 48 h) decreases the expressions of PARP, caspase 3, and 9[1].
Bromosporine (0.1, 0.5 and 1 μM; 6-10 days) inhibits AML cells in a dose-dependent manner[3].
Bromosporine (2.5 μM; 72 h) activates HIV-1 replication in vitro in latent HIV-1 J-Lat clone C11 cells[4].
Bromosporine (1-50 μM; 48 h) does not induce marked toxicity in primary CD4+ T cells[4].
MCE has not independently confirmed the accuracy of these methods. They are for reference only.

Cell Proliferation Assay[1]

Cell Line:	HCT116 and HT29
Concentration:	0, 30, 60, 120, 240, 480 and 1000 nM
Incubation Time:	72 h
Result:	Synergistically inhibited cell growth in CRC cells with 5-FU (HY-90006) (0-16 μg/mL) and exhibited a dose-dependent manner.

Cell Cycle Analysis[1]

Cell Line:	HCT116 and HT29
Concentration:	Various concentration
Incubation Time:	48 h
Result:	Caused a distinct increase in the cells arrested at G1 phase when combined with 5-FU (HY-90006).

Western Blot Analysis[1]

Cell Line:	HCT116 and HT29
Concentration:	Various concentration
Incubation Time:	48 h
Result:	Elevated the level of apoptosis in both cell lines through cleavage of PARP, caspase 3, and 9.

Cell Proliferation Assay[3]

Cell Line:	MV4;11, KASUMI-1, OCI-AML3 and K562
Concentration:	0.1, 0.5 and 1 μM
Incubation Time:	6-10 days
Result:	Inhibited these AML cells in a dose-dependent manner.

Cell Cytotoxicity Assay[4]

Cell Line:	PBMCs
Concentration:	1 μM, 2.5 μM, 5 μM, 10 μM, 25 μM and 50 μM
Incubation Time:	48 h
Result:	Did not induce marked toxicity in primary CD4+ T cells with CC_{50} over 10 μM.

In Vivo

Bromosporine (100 mg/kg; i.p.; daily for 10 days) shows better antitumor activity than individual when co-treated with 5-FU (HY-90006)[1].

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

Animal Model:	Female BALB/c nude mice (5-6 weeks; injected with 1×10^6 cells/100 μL of HT116 cells)[1]
Dosage:	100 mg/kg
Administration:	i.p.; daily for 10 days
Result:	Exhibited better antitumor activity than individual Bromosporine or 5-FU (HY-90006) when co-treated with the two agent.

CUSTOMER VALIDATION

- Proc Natl Acad Sci U S A. 2019 Feb 19;116(8):2961-2966.
- Biochem Pharmacol. 2020 Jul;177:113946.
- Patent. US20180263995A1.

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REFERENCES

[1]. Cheng X, et al. BET inhibitor bromosporine enhances 5-FU effect in colorectal cancer cells. Biochem Biophys Res Commun. 2020 Jan 22;521(4):840-845.

[2]. El-Shershaby MH, et al. From triazolophthalazines to triazoloquinazolines: A bioisosterism-guided approach toward the identification of novel PCAF inhibitors with potential anticancer activity. Bioorg Med Chem. 2021 Jul 15;42:116266.

[3]. Picaud S, et al. Promiscuous targeting of bromodomains by bromosporine identifies BET proteins as master regulators of primary transcription response in leukemia. Sci Adv. 2016 Oct 12;2(10):e1600760.

[4]. Pan H, et al. The bromodomain and extraterminal domain inhibitor bromosporine synergistically reactivates latent HIV-1 in latently infected cells. Oncotarget. 2017 Oct 6;8(55):94104-94116.

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